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High-Level and Transuranic Radioactive Wastes

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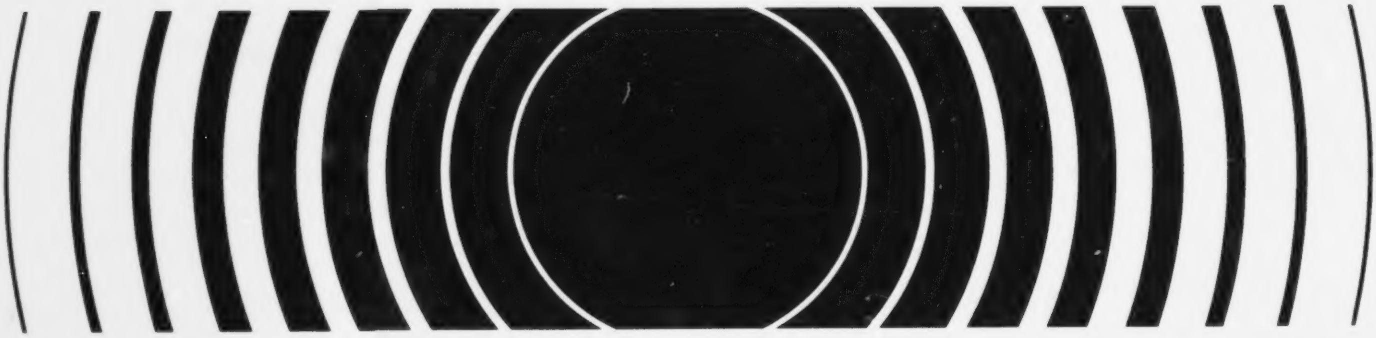

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This document provides background information that, when considered together with the promulgated generally applicable standards, supports the final actions taken by the EPA with regard to the management and disposal of spent nuclear fuel and high-level and transuranic wastes. It also contains an integrated risk assessment that provides a scientific basis for these actions.

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BACKGROUND INFORMATION DOCUMENT
FINAL RULE FOR HIGH-LEVEL AND TRANSURANIC RADIOACTIVE WASTES

August 1985

U.S. Environmental Protection Agency
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Chapter 1: INTRODUCTION

The U.S. Environmental Protection Agency (EPA) is responsible for developing and issuing environmental standards, guidelines, and criteria to ensure that the public and the environment are adequately protected from potential radiation impacts.

Toward this end, EPA is promulgating generally applicable environmental standards for the management and disposal of spent nuclear fuel and high-level and transuranic radioactive wastes. These standards provide the basic framework for long-term control through management and disposal of three types of waste:

1) Spent nuclear reactor fuel if disposed of without reprocessing.

2) High-level radioactive liquid or solid wastes from the reprocessing of spent nuclear fuel.

3) Transuranic wastes containing long-lived radionuclides of elements heavier than uranium and defined as containing 100 nanocuries or more of alpha-emitting transuranic nuclides, with half-lives greater than 20 years, per gram of waste.

1.1 EPA Authorities for the Rulemaking

These standards have been developed pursuant to the Agency's authorities under the Atomic Energy Act of 1954, as amended, and Reorganization Plan No. 3 of 1970.

The basic authority under the Atomic Energy Act of 1954, as amended and transferred from the Atomic Energy Commission to EPA through the Reorganization Plan No. 3 of 1970, includes the function of "establishing generally applicable environmental standards for the protection of the general environment from radioactive material. As used herein, standards mean limits on radiation exposures or levels, or concentrations or quantities of radioactive material, in the general environment outside the boundaries of locations under the control of persons possessing or using radioactive material" (Ni70).

The Nuclear Waste Policy Act (NWPA) of 1982 established formal procedures regarding the evaluation and selection of sites for geologic repositories, including procedures for the interaction of State and

Federal governments; reiterated the existing responsibilities of the Federal agencies involved in the national program; and provided a timetable for several key milestones to be met by the Federal agencies in carrying out the program. As part of this national program, the EPA, pursuant to its authorities under other provisions of law, "shall, by rule, promulgate generally applicable standards for protection of the general environment from offsite releases from radioactive material in repositories" (NWPA82).

1.2 History of the High-Level Radioactive Waste Program and the EPA Rulemaking

Since the inception of the nuclear age in the 1940's, the Federal government has assumed ultimate responsibility for the care and disposal of high-level radioactive wastes regardless of whether they are produced by commercial or national defense activities. In 1949 the Atomic Energy Commission (AEC) initiated research and development work aimed at developing systems for the conversion of high-level liquid wastes to chemically stable solids. Then, in 1955, at the request of the AEC, a National Academy of Sciences - National Research Council (NAS-NRC) advisory committee was established to consider the disposal of high-level radioactive wastes within the United States. Their report, issued in 1957, contained two general recommendations (NAS57): 1) that the AEC continue efforts to develop processes for the solidification of high-level radioactive liquid wastes, and 2) that naturally occurring salt formations are the most promising medium for the long-term isolation of these solidified wastes. Project Salt Vault, conducted from 1965-1967 by the AEC in an abandoned salt mine near Lyons, Kansas, demonstrated the safety and feasibility of handling and storing solid wastes in salt formations (Mc70).

In 1968, the AEC again requested the NAS-NRC to establish a Committee on Radioactive Waste Management (CRWM) to advise the AEC concerning its long-range radioactive waste management plans and to evaluate the feasibility of disposing of solidified radioactive wastes in bedded salt. The CRWM convened a panel to discuss the disposal of radioactive waste in salt mines. Based on the recommendations of the panel, the CRWM concluded that the use of bedded salt is satisfactory for the disposal of radioactive waste (NAS70).

In 1970, the AEC announced the tentative selection of a site at Lyons, Kansas, for the establishment of a national radioactive waste repository (AEC70). During the next two years, however, in-depth site studies raised several questions concerning the safe plugging of old exploratory wells and proposed expanded salt mining activities. These questions and the growing public opposition to the Lyons site prompted the AEC in late 1971 to pursue alternatives to the salt site at Lyons (Do72).

In 1976, the Federal government intensified its program to develop and demonstrate a permanent disposal method for high-level radioactive wastes. The Office of Management and Budget (OMB) established an interagency task force on commercial nuclear wastes in March 1976. The OMB interagency task force defined the scope and responsibility of each Federal agency's activities on high-level waste management, including the preparation of environmental standards for high-level waste by EPA (Ly76, En77a,b).

A status report on the management of commercial radioactive nuclear wastes, published in May 1976 by the President's Federal Energy Resources Council (ERC), emphasized the need for coordination of administration policies and programs relating to energy. The ERC established a nuclear subcommittee to coordinate Federal nuclear policy and programs to assure an integrated government effort. This report called for an accelerated comprehensive government radioactive waste program plan calling for an interagency task force to coordinate activities among the responsible Federal agencies. The EPA was given the responsibility of establishing general environmental standards governing waste activities, including high-level radioactive wastes that must be delivered to Federal repositories for long-term management (FERC76).

In October 1976, President Ford issued a major statement on nuclear policy. As part of his comprehensive statement, he announced new steps to assure that the United States has the facilities for long-term management of nuclear wastes from commercial powerplants. The President's actions were based on the findings of the OMB interagency task force formed in March 1976. He announced that the experts had concluded that the most practical method for disposing of high-level waste is geologic storage in repositories in stable formations deep underground. Among the many steps to be taken was EPA's issuance of general environmental standards governing nuclear facility releases to the biosphere above the natural background radiation level (Fo76). These standards were to place a numerical limit on long-term radiation releases outside the boundary of the repository.

In December 1976, EPA announced its intent to develop environmental radiation protection standards for high-level radioactive waste to assure protection of the public health and the general environment (EPA76). These efforts have included frequent interaction with the public, which began with a series of public workshops on radioactive waste disposal in 1977 and 1978 (EPA77a,b, EPA78a,b).

In 1978, President Carter established the Interagency Review Group (IRG) to develop recommendations for the establishment of an administrative policy with respect to long-term management of nuclear wastes and supporting programs to implement the policy. The IRG report reemphasized EPA's role in developing generally applicable standards for the disposal of high-level waste, spent nuclear fuel, and transuranic wastes (DOE79). In a Message to Congress on February 12, 1980, the President outlined a comprehensive national radioactive waste management program based on the IRG report. The message called for an interim strategy for disposal of high-level and transuranic wastes that would rely on mined-out geologic repositories. The message repeated that the EPA was responsible for creating general criteria and numerical standards applicable to nuclear waste management activities (Ca80).

In November 1978, the EPA published proposed "Criteria for Radioactive Wastes," which were intended as Federal guidance for storage and disposal of all forms of radioactive wastes (EPA78c). In March 1981, however, EPA withdrew the proposed criteria because the many different types of radioactive wastes made the issuance of generic disposal guidance too difficult (EPA81).

Development efforts continued, and on December 29, 1982, EPA published a proposed rule on "Environmental Radiation Protection Standards for the Management and Disposal of Spent Nuclear Fuel, High-Level and Transuranic Radioactive Wastes" (EPA82).

Shortly after the EPA proposed rule was published, Congress passed the Nuclear Waste Policy Act of 1982 (P.L. 97-425), wherein the EPA was to "...promulgate generally applicable standards for protection of the general environment from offsite releases from radioactive material in repositories" not later than January 1984 (NWPA83).

After the first comment period on the proposed rule ended on May 2, 1983, EPA held two public hearings on the proposed standards--one in Washington, D.C., May 12-14, 1983, and one in Denver, Colorado, May 19-21, 1983--and requested post-hearing comments during a second comment period (EPA83a,b). More than 200 comment letters were received during these two comment periods, and 13 oral statements were made at the public hearings. Responses to comments received from the public are presented in "Response to Comments, Volume I - Public Comments" (a companion document).

In parallel with this public review and comment, the Agency conducted an independent scientific review of the technical basis for the proposed 40 CFR 191 standards through a special Subcommittee of the Agency's Science Advisory Board (SAB). This Subcommittee held nine public meetings from January 18, 1983, through September 21, 1983 (EPA83c). The SAB then prepared a final report that was transmitted on February 17, 1984 (SAB84). Although the SAB review found that the Agency's analyses in support of the proposed standards were comprehensive and scientifically competent, the report contained several findings and recommendations for improvement. The public was notified of the availability of this report on May 8, 1984, and encouraged to comment on the findings and recommendations (EPA84). Responses to the SAB report are presented in another companion document entitled "Response to Comments, Volume II - Science Advisory Board Comments."

On February 8, 1985, the Natural Resources Defense Council, Inc., the Environmental Defense Fund, the Environmental Policy Institute, the Sierra Club, and the Snake River Alliance brought suit against the Agency and the Administrator because of noncompliance with the January 7, 1984, deadline mandated by the NWPA for promulgation of standards. A consent order was negotiated with the plaintiffs that required the standards to be promulgated on or before August 15, 1985.

1.3 Purpose and Scope of BID

~~The purpose of~~ this document ~~is to~~ provides background information that, when considered together with the promulgated generally applicable standards, supports the final actions taken by the EPA with regard to the management and disposal of spent nuclear fuel and high-level and transuranic wastes. It also contains an integrated risk assessment that provides a scientific basis for these actions.

The scope encompasses the conceptual framework for assessing radiation risks, including identification of the sources of possible radionuclide releases, analysis of the movement of the radionuclides from the source through environmental pathways, estimates of doses received by human individuals and populations, and calculations of the probability of genetic and somatic health effects.

1.4 Computer Codes Utilized

A number of computer codes have been used as tools in the Agency's risk analyses. The central tool has been the program REPRISK, which has been under development at the Agency since 1978. This code, described in Chapter 8, makes use of conversion factors that relate the amount of radioactive material released to the accessible environment to population health effects. These conversion factors are obtained by using another EPA computer code called WESPDOSE (Sm85). WESPDOSE considers a number of pathways for the environmental transport of radionuclides. For calculations involving individual doses and time frames longer than ten thousand years, the computer code NWFT/ DVM, developed by Sandia under contract to NRC, has been used (Ca81). This code models the transport of decay chains whose elements have different retardation factors in the groundwater system. A more complex groundwater code, SWIFT, has also been used to support the EPA risk analyses, primarily to validate some of the hydrologic calculations carried out using simpler models (Re81).

Four kinds of "release mechanisms" are addressed by REPRISK:

1) Direct impact on a waste package with associated releases to the air and/or the land surface (e.g., volcano, meteorite, drilling/direct hit).

2) Direct impact on a waste package with associated releases to an aquifer (e.g., faulting, breccia pipes).

3) Disruption of the repository with associated releases to the land surface (e.g., drilling/no hit).

4) Disruption of the repository with associated releases to an aquifer (e.g., normal groundwater flow, faulting, breccia pipes, drilling/no hit).

Each release mechanism leads to several pathways to human exposure. The consequences of a radioactivity release to the accessible environment are expressed in terms of 1) number of somatic health effects (fatal cancers), 2) number of genetic health effects, 3) ratio of released amount to the release limit in 40 CFR Part 191, and/or 4) curies released of each radionuclide.

Two time frames are used by the model. One, called a dose commitment period, is for modeling the occurrence of release mechanisms at the site. The other, a dose integration period, is used for measuring the consequences of the releases. This way, consequences may be measured beyond the time when a particular perturbation may be active at a site.

1.5 Program Technical Support Documents

A number of technical support documents have been prepared and published during the history of the rulemaking and standards program to help establish the technical basis for the standards. These documents should also be considered as part of the technical background for the present rulemaking process. The following is a listing of these documents and a short abstract of each.

(1) Technical Support of Standards for High-Level Radioactive Waste Management - Volume A, Source Term Characterization, EPA 520/4-79-007A, March-July 1977.

This report provides a characterization of commercial spent nuclear fuel and high-level waste, including comparisons of source terms from various fuel cycles and fuel mixes; a characterization of government high-level and transuranic wastes; a comparison with commercial waste; and an estimation of existing and projected quantities of spent nuclear fuel and high-level and transuranic wastes. The data are presented in several formats and on a specific basis (per unit of fuel used or energy generated), as well as on a total basis for a given number of nuclear powerplants.

(2) Technical Support of Standards for High-Level Radioactive Wastes Management - Volume B, Engineering Controls, EPA 520/4-79-007B, March-August 1977.

This report reviews the technology for engineering control of spent fuel and high-level and TRU wastes and projected costs of the various disposal technologies. Analysis includes processing and packaging technology, alternative geologic disposal techniques, effectiveness of engineering controls, and the cost considerations.

(3) Technical Support of Standards for High-Level Radioactive Waste Management - Volume C, Migration Pathways, EPA 520/4-79-007C, March-July 1977.

This report assesses geologic site selection factors; quantifies the potential for the migration of nuclides through the geosphere to the biosphere, and considers dose implications of a repository for wastes containing large quantities of radionuclides in high concentrations that might become dispersed into the biosphere over geologic times.

(4) Technical Support of Standards for High-Level Radioactive Waste Management - Volume D, Release Mechanisms, EPA 520/4-79-007D, March 1980.

This report analyzes the potential for the release of radionuclides from a generic deep-mined repository for radioactive wastes. Five different geologic media are considered: bedded salt, dome salt, granite, basalt, and shale. A range of potential containment failure mechanisms were evaluated and compared. Results are combined with radionuclide transport and dose calculations for assessment of the potential effects of a repository on human health.

(5) Technical Support of Standards for High-Level Radioactive Waste Management - Addendum to Volumes C and D, EPA 520/4-79-007E, March 1982.

This report is an update of information and issues relevant to the conclusions of Volumes C and D.

(6) Assessment of Waste Management of Volatile Radionuclides, EPA ORP/CSD-79-2, May 1979.

This report reviews waste management technologies in terms of immobilization, containment, and disposal of the radionuclides I-129, Kr-85, H-3, and C-14. Included are alternative disposal options that may be applied to isolate these wastes from the human environment.

(7) Radiation Exposures From Solidification Processes for High-Level Radioactive Liquid Wastes, EPA 520/3-80-007, May 1980.

This report is an assessment of a generic high-level liquid waste solidification plant and the potential environmental impact of atmospheric discharges during normal operations involving four different solidification processes.

(8) A Review of Radiation Exposure Estimates From Normal Operations in the Management and Disposal of High-Level Radioactive Wastes and Spent Nuclear Fuel, EPA 520/3-80-008, August 1980.

This report provides an analysis of the estimated radioactive releases during normal waste management operations (i.e., preparation for storage or disposal, storage, and emplacement) and the resulting radiation doses.

(9) Alternative Disposal Concepts for High-Level and Transuranic Radioactive Waste Disposal, EPA ORP/CSD-79-1, May 1979.

This report examines several technologies that have been proposed as alternative concepts to geologic disposal, including transmutation, extraterrestrial disposal, seabed disposal, ice-sheet disposal, and other continental geologic disposal.

(10) Economic Impacts of 40 CFR 191: Environmental Standards and Federal Guidance for Management and Disposal of Spent Nuclear Fuel, High-Level and Transuranic Radioactive Wastes, EPA 520/4-80-014, December 1980.

This report develops a methodology for examining the potential economic impacts of the proposed environmental standards.

(11) Environmental Pathway Models for Estimating Population Health Effects from Disposal of High-Level Radioactive Waste in Geologic Repositories, Draft Report EPA 520/5-80-002, December 1982.

This report describes the mathematical models formulated to calculate the environmental dose commitments and population health effects (fatal cancers and first generation genetic defects) that could occur as a result of releases from geologic repositories.

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Chapter 2: CURRENT REGULATORY PROGRAMS AND STRATEGIES

2.1 Introduction

People have always been exposed to ionizing radiations from cosmic rays and the naturally-occurring radionuclides in the earth that make up the natural radiation background. Awareness of radiation and radioactivity dates back only to the end of the last century--to the discovery of x-rays in 1895 and the discovery of radioactivity in 1896. These discoveries marked the beginning of radiation science and the deliberate use of radiation and radionuclides in science, medicine, and industry.

The findings of radiation science rapidly led to the development of medical and industrial radiology, nuclear physics, and nuclear medicine. By the 1920's, the use of x-rays in diagnostic medicine and industrial applications was widespread, and radium was being used by industry for luminescent dials and by doctors in therapeutic procedures. By the 1930's, biomedical and genetic researchers were studying the effects of radiation on living organisms, and physicists were beginning to understand the mechanisms of spontaneous fission and radioactive decay. By the 1940's, a self-sustaining fission reaction was demonstrated, which led directly to the construction of the first nuclear reactors and atomic weapons.

Developments since the end of World War II have been rapid. Today the use of x-rays and radioactive materials is widespread and includes:

- Nuclear reactors, and their supporting fuel-cycle facilities, which generate electricity and power ships and submarines; produce radioisotopes for research, space, defense, and medical applications; and are used as research tools for nuclear engineers and physicists.

- Particle accelerators which produce radioisotopes and are used as research tools for studying the structure of materials and atoms.

- The radiopharmaceutical industry which provides the radioisotopes needed for biomedical research and nuclear medicine.

- Nuclear medicine which has developed as a recognized medical specialty in which radioisotopes are used in the diagnosis and treatment of numerous diseases.

- X-rays which are widely used as a diagnostic tool in medicine and in such diverse industrial fields as oil exploration and nondestructive testing.

- Radionuclides which are used in such common consumer products as luminous-dial wristwatches and smoke detectors.

The following sections of this chapter provide a brief history of the evolution of radiation protection philosophy and an outline of the current regulatory programs and strategies of the government agencies responsible for assuring that radiation and radionuclides are used safely.

2.2 The International Commission on Radiological Protection and the National Council on Radiation Protection and Measurements

Initially, the dangers and risks posed by x-rays and radioactivity were poorly understood. By 1896, however, "x-ray burns" were being reported in the medical literature, and by 1910, it was understood that such "burns" could be caused by radioactive materials. By the 1920's, sufficient direct evidence (from the experiences of radium dial painters, medical radiologists, and miners) and indirect evidence (from biomedical and genetic experiments with animals) had been accumulated to persuade the scientific community that an official body should be established to make recommendations concerning human protection against exposure to x-rays and radium.

At the Second International Congress of Radiology meeting in Stockholm, Sweden, in 1928, the first radiation protection commission was created. Reflecting the uses of radiation and radioactive materials at the time, the body was named the International X-Ray and Radium Protection Commission and was charged with developing recommendations concerning protection from radiation. In 1950, to reflect better its role in a changing world, the commission was reconstituted and renamed the International Commission on Radiation Protection (ICRP).

During the Second International Congress of Radiology, the newly created commission suggested to the nations represented at the Congress that they appoint national advisory committees to represent their viewpoints before the ICRP, and to act in concert with the Commission in developing and disseminating recommendations on radiation protection. This suggestion led to the formation, in 1929, of the Advisory Committee on X-Ray and Radium Protection as the U.S. advisory group. This Advisory Committee, after a series of reorganizations and name changes, emerged in 1964 in its present form as the Congressionally-chartered

National Council on Radiation Protection and Measurements (NCRP). The Congressional charter provides for the NCRP to:

- Collect, analyze, develop, and disseminate in the public interest information and recommendations about radiation protection and radiation quantities, units, and measurements.
- Develop basic concepts about radiation protection and radiation quantities, units, and measurements, and the application of these concepts.
- Provide a means by which organizations concerned with radiation protection and radiation quantities, units, and measurements may cooperate to effectively use their combined resources, and to stimulate the work of such organizations.
- Cooperate with the ICRP and other national and international organizations concerned with radiation protection and radiation quantities, units, and measurements.

Throughout their existence, the ICRP and the NCRP have worked together closely to develop radiation protection recommendations that reflect the current understanding of the dangers associated with exposure to ionizing radiation.

The first exposure limits adopted by the ICRP and the NCRP (ICRP34, ICRP38, NCRP36) established 0.2 roentgen/day* as the "tolerance dose" for occupational exposure to x-rays and gamma radiation from radium. This limit, equivalent to approximately 25 rads/year as measured in air, was established to guard against the known effects of ionizing radiation on superficial tissue, changes in the blood, and "derangement" of internal organs, especially the reproductive organs. At the time the recommendations were made, high doses of radiation were known to cause observable effects and even to induce cancer. However, no such effects were observed at lower doses, and the epidemiological evidence at the time was inadequate to even imply the carcinogenic induction effects of moderate or low doses. Therefore, the aim of radiation protection was to guard against known effects, and the "tolerance dose" limits that were adopted were believed to represent the level of radiation that a person in normal health could tolerate without suffering observable effects. The concept of a tolerance dose and the recommended occupational exposure limit of 0.2 R/day for x- and gamma radiation remained in effect until the end of the 1940's. The recommendations of the ICRP and the NCRP made no mention of exposure of the general populace.

By the end of World War II, the widespread use of radioactive materials and scientific evidence of genetic and somatic effects at lower doses and dose rates suggested that the radiation protection recommendations of the NCRP and the ICRP would have to be revised downward.

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* The NCRP's recommendation was 0.1 roentgen(R)/day measured in air. This limit is roughly equivalent to the ICRP limit, which was conventionally measured at the point of exposure and included back-scatter.

By 1948, the NCRP had formulated its position on appropriate new limits. These limits were largely accepted by the ICRP in its recommendations of 1950 and formally issued by the NCRP in 1954 (ICRP51, NCRP54). The immediate effect was to lower the basic whole-body occupational dose limit to 0.3 rad/week (approximately 15 rads/year); the revised recommendations also embodied several new and important concepts in the formulation of radiation protection criteria.

First, the recommendations recognized the differences in the effects of various types and energies of radiation; both ICRP's and NCRP's recommendations included discussions of the weighting factors that should be applied to radiations of differing types and energies. The NCRP advocated the use of the "rem" to express the equivalence in biological effects between radiations of differing types and energy.* Although the ICRP noted the shift toward the acceptance of the rem, it continued to express its recommendations in terms of the rad, with the caveat that neutrons should carry a quality factor of ten.

Second, the recommendations of both organizations introduced the concept of critical organs and tissues. The intent of this concept was to assure that no tissue or organ, with the exception of the skin, would receive a dose in excess of that allowed for the whole body. At the time, scientific evidence was lacking on which to base different recommended limits for the various tissues and organs. Thus, all blood-forming organs were considered critical organs and were limited to the same exposure as the whole body. The skin was allowed an exposure of 30 rads/year and the extremities were allowed a limit of 75 rads/year.

Third, the recommendations of the NCRP included the suggestion that individuals under the age of 18 receive no more than one-tenth the exposure allowed for adults. The reasoning behind this particular recommendation is interesting as it reflects clearly the limited knowledge of the times. The scientific evidence indicated a clear relationship between accumulated dose and genetic effect. However, this evidence was obtained exclusively from animal studies that had been conducted with doses ranging from 25 to thousands of rads. There was no evidence from exposures less than 25 rads accumulated dose, and the interpretation of the animal data and the implications for humans was unclear and did not support a specific permissible dose. The data did suggest that genetic damage was more dependent on accumulated dose than previously believed, but experience showed that exposure for prolonged periods to the permissible dose (1.0 R/week) did not result in any observable genetic effects. The NCRP decided that it was not necessary to change the occupational limit to provide additional protection beyond that provided by the reduction in the permissible dose limit to 0.3 R/week. At the same time, it recommended limiting the exposure of individuals under the age of 18 to assure that they did not accumulate a genetic dose that would later preclude their employment as radiation workers. The factor of ten was rather arbitrary, but was believed to be sufficient to protect the future employability of all individuals (NCRP54).

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* The exact relationship between roentgens, rads, and rems is beyond the scope of this work. In simple terms, the roentgen is a measure of the degree of ionization induced by x- and gamma radiations in air. The rad (radiation absorbed dose) is a measure of the energy imparted to matter by radiation. And the rem (roentgen equivalent man) is a measure of equivalence for the relative biological effect of radiations of different types and energies on man. Over the range of energies typically encountered, the relationship of roentgens to rads to rems for x- and gamma radiation is essentially equality. For beta radiation, rads are equivalent to rems, and for alpha radiation one rad equals 10 to 20 rems.

Fourth, the concept of a tolerance dose was replaced by the concept of a maximum permissible dose. The change in terminology reflected the increasing awareness that any radiation exposure might involve some risk and that repair mechanisms might be less effective than previously believed. Therefore, the concept of a maximum permissible dose was adopted because it better reflects the uncertainty in our knowledge than does the concept of tolerance dose. The maximum permissible dose was defined as the level of exposure that entailed a small risk compared with those posed by other hazards in life (ICRP51).

Finally, in explicit recognition of the inadequacy of our knowledge regarding the effects of radiation and of the possibility that any exposure might have some potential for harm, the recommendations included an admonition that every effort should be made to reduce exposure to all kinds of ionizing radiation to the lowest possible level. This concept, known originally as ALAP (as low as practicable) and later as ALARA (as low as reasonably achievable), would become a cornerstone of radiation protection philosophy.

During the 1950's, a great deal of scientific evidence on the effects of radiation became available from studies of the radium dial painters, radiologists, and the survivors of the atomic bombs dropped on Japan. This evidence suggested that genetic effects and long-term somatic effects were more important than previously considered. Thus, by the late 1950's, the ICRP and NCRP recommendations were again revised (ICRP59, NCRP59). These revisions included the following major changes: the annual maximum permissible dose for whole-body exposure and the most critical organs (blood-forming organs, gonads, and the lens of the eye) was lowered to 5 rems, with a quarterly limit of 3 rems; the limit for exposure of other organs was set at 30 rems/year; internal exposures were controlled by a comprehensive set of maximum permissible concentrations of radionuclides in air and water based on the most restrictive case of a young worker; and recommendations were included for some nonoccupational groups and for the general population (for the first time).

The lowering of the annual maximum permissible whole-body dose to 5 rems, with a quarterly limit of 3 rems, reflects both the new evidence and the uncertainties of the time. Although no adverse effects were observed among workers who had received the earlier maximum permissible dose of 0.3 rad in a week, there was concern that the lifetime accumulation of as much as 750 rads (15 rads/year times 50 years) was too much. Lowering the maximum permissible dose by a factor of three was believed to provide a greater margin of safety. At the same time, operational experience showed that an annual dose of 5 rems could be met in most instances, particularly with the additional operational flexibility provided by expressing the limit on an annual and quarterly basis.

The recommendations given for nonoccupational exposures were based on concerns of genetic effects. The evidence available suggested that genetic effects were primarily dependent on the total accumulated dose. Thus, having sought the opinions of respected geneticists, the ICRP and the NCRP adopted the recommendation that accumulated gonadal dose to age 30 be limited to 5 rems from sources other than natural background and medical exposure. As an operational guide, the NCRP recommended that the maximum annual dose to any individual be limited to 0.5 rem, with maximum permissible body burdens of radionuclides (to control internal exposures) set at one-tenth that allowed for radiation workers. These values were derived from consideration of the genetically significant dose to the population, and were established "primarily for the purpose of keeping the average dose to the whole population as low as reasonably possible, and not because of the likelihood of specific injury to the individual" (NCRP59).

During the 1960's, the ICRP and NCRP again lowered the maximum permissible dose limits (ICRP65, NCRP71). The considerable scientific data on the effects of exposure to ionizing radiation were still inconclusive with respect to the dose-response relationship at low exposure levels; thus, both organizations continued to stress the need to keep all exposures to the lowest possible level.

The NCRP and the ICRP made the following similar recommendations:

- Limit the dose to the whole-body, red bone marrow, and gonads to 5 rems in any year, with a retrospective limit of 10 to 15 rems in any given year as long as total accumulated dose did not exceed 5x(N-18), where N is age in years.
- Limit the annual dose to the skin, hands, and forearms to 15, 75, and 30 rems, respectively.
- Limit the annual dose to any other organ or tissue to 15 rems.
- Limit the annual dose to any nonoccupationally exposed individual in the population to 0.5 rem.
- Limit the annual average dose to the population to 0.17 rem.

The scientific evidence and the protection philosophy on which the above recommendations were based were set forth in detail in NCRP71. In the case of occupational exposure limits, the goal of protection was to ensure that the risks of genetic and somatic effects were small enough to be comparable to the risks experienced by workers in other industries. The conservatively derived numerical limits recommended were based on the linear, nonthreshold, dose-response model, and were believed to represent a level of risk that was readily acceptable to an average individual. For nonoccupational exposures, the goal of protection was to ensure that the risks of genetic or somatic effects were small compared with other risks encountered in everyday life. The derivation of specific limits was complicated by the unknown dose-response relationship at low exposure levels and the fact that the risks of radiation exposure did not necessarily accrue to the same individuals who benefited from the activity responsible for the exposure. Therefore, it was necessary to derive limits that gave adequate protection to each member of the public and to the gene pool of the population as a whole, while still allowing the development of beneficial uses of radiation and radionuclides.

In 1977, the ICRP made a fundamental change in its recommendations when it abandoned the critical organ concept in favor of the weighted whole-body dose equivalent concept for limiting occupational exposure (ICRP77). The change, made to reflect our increased understanding of the differing radiosensitivity of the various organs and tissues, did not affect the overall limit of 5 rems per year and is not intended to be applied to nonoccupational exposures.

Also significant is the fact that ICRP's 1977 recommendations represent the first explicit attempt to relate and justify permissible radiation exposures with quantitative levels of acceptable risk. Thus, the risks of average occupational exposures (approximately 0.5 rem/year) are equated with risks in safe industries, given as 10^{-4} annually. At the maximum limit of 5 rems/year, the risk is equated with that experienced by some workers in recognized hazardous occupations. Similarly, the risks implied by the nonoccupational limit of 0.5 rem/year are equated to levels of risk of less than 10^{-2} in a lifetime; the general populace's average exposure is equivalent to a lifetime risk on the order of 10^{-3} to 10^{-4}. The ICRP believed these levels of risk were in the range that most individuals find acceptable.

The NCRP has not formally changed its recommendations for occupational exposure to correspond to the 1977 recommendations of the ICRP. It has been working diligently, however, to review its recommendations, and has circulated a draft of proposed changes to various interested scientists and regulatory bodies for their comment. The relevant nonoccupational exposure limits are:

- 0.5 rem/year whole-body dose equivalent, not including background or medical radiation, for individuals in the population when the exposure is not continuous.

- 0.1 rem/year whole-body dose equivalent, not including background or medical radiation, for individuals in the population when the exposure is continuous.

- Continued use of a total dose limitation system based on justification of every exposure and application of the ALARA philosophy to every exposure.

The NCRP equates continuous exposure at the level of 0.1 rem/year to a lifetime risk of developing cancer of about one in a thousand. The NCRP has not formulated exposure limits for specific organs, but it notes that the permissible limits will necessarily be higher than the whole-body limit in inverse ratio of the risk for a particular organ to the total risk for whole-body exposure.

2.3 Federal Guidance

The ICRP and the NCRP function as nongovernmental advisory bodies. Their recommendations are not binding on any user of radiation or radioactive materials. The wealth of new scientific information on the effects of radiation that became available in the 1950's prompted President Eisenhower to establish an official government entity with responsibility for formulating radiation protection criteria and coordinating radiation protection activities. Thus, the Federal Radiation Council (FRC) was established in 1959 by Executive Order 10831. The Council included representatives from all of the Federal agencies concerned with radiation protection, and acted as a coordinating body for all of the radiation activities conducted by the Federal government. In addition to its coordinating function, the Council's major responsibility was to "...advise the President with respect to radiation matters, directly or indirectly affecting health, including guidance for all Federal agencies in the formulation of radiation standards and in the establishment and execution of programs of cooperation with States..." (FRC60).

The Council's first recommendations concerning radiation protection standards for Federal agencies were approved by the President in 1960. Based largely on the work and recommendations of the ICRP and the NCRP, the guidance established the following limits for occupational exposures:

- Whole body, head and trunk, active blood forming organs, gonads, or lens of eye--not to exceed 3 rems in 13 weeks and total accumulated dose limited to 5 times the number of years beyond age 18.

- Skin of whole body and thyroid--not to exceed 10 rems in 13 weeks or 30 rems per year.

- Hands, forearms, feet, and ankles--not to exceed 25 rems in 13 weeks or 75 rems per year.

- Bone--not to exceed 0.1 microgram of radium-226 or its biological equivalent.

- Any other organ--not to exceed 5 rems per 13 weeks or 15 rems per year.

Although these levels differ slightly from those recommended by NCRP and ICRP at the time, the differences do not represent any greater or lesser protection. In fact, the FRC not only accepted the levels recommended by the NCRP for occupational exposure, it adopted the NCRP's philosophy of acceptable risk for determining occupational exposure limits. Although quantitative measures of risk were not given in the guidance, the prescribed levels were not expected to cause appreciable bodily injury to an individual during his or her lifetime. Thus, while the possibility of some injury was not zero, it was so low as to be acceptable if there was any significant benefit derived from the exposure.

The guidance also established exposure limits for members of the public. These were set at 0.5 rem per year (whole body) for an individual, and an average of 5 rems in 30 years (gonadal) per capita. The guidance also provided for developing a suitable sample of the population as an operational basis for determining compliance with the limit when doses to all individuals are unknown. Exposure to this population sample was not to exceed 0.17 rem per capita per year. The population limit of 0.5 rem to any individual per year, was derived from consideration of natural background exposure.

In addition to the formal exposure limits, the guidance also established as Federal policy that there should be no radiation exposure without an expectation of benefit, and that "every effort should be made to encourage the maintenance of radiation doses as far below this guide as practicable." The inclusion of the requirements to consider benefits and keep all exposure to a minimum was based on the possibility that there is no threshold dose for radiation. The linear nonthreshold dose response was assumed to place an upper limit on the estimate of radiation risk. However, the FRC explicitly recognized that it might also represent the true level of risk. If so, then any radiation exposure carried some risk, and it was necessary to avoid all unproductive exposures and to keep all productive exposures as "far below this guide as practicable."

In 1967, the Federal Radiation Council issued guidance for the control of radiation hazards in uranium mining (FRC67). The need for such guidance was clearly indicated by the epidemiological evidence that showed a higher incidence of lung cancer in adult males who worked in uranium mines compared with the incidence in adult males from the same locations who had not worked in mines. The guidance established specific exposure limits and recommended that all exposures be kept as far below the guide limits as possible. The limits chosen represented a trade-off between the risks incurred at various exposure levels, the technical feasibility of reducing the exposure, and the benefits of the activity responsible for the exposure. The guidance also applied to nonuranium mines.

In 1970, the functions of the Federal Radiation Council were transferred to the U.S. Environmental Protection Agency (EPA). In 1971, the EPA revised the Federal guidance for the control of radiation hazards in underground uranium mining (EPA71). Based on the risk levels associated with the exposure limits established in 1967, the upper limit of exposure was reduced by a factor of three. The EPA has also provided federal guidance for the diagnostic use of x-rays (EPA78). This guidance established maximum skin entrance doses for various types of routine x-ray examinations. It also established the requirement that all x-ray exposures be based on clinical indication and diagnostic need, and that all exposure of patients should be kept as low as reasonably achievable consistent with the diagnostic need.

In 1981, the EPA proposed new Federal guidance for occupational exposures to supersede the 1960 guidance (EPA81). The 1981 recommended guidance follows the principles set forth by the ICRP in 1977, with respect to combining internal and external doses. The basic occupational limit suggested in the guidance is 5 rems per year. This recommended guidance has not yet been adopted as Federal policy. The proposals in the guidance were issued for public comment in 1981 and are currently being reviewed and revised in light of the comments received.

2.4 The Environmental Protection Agency

In addition to the statutory responsibility to provide Federal guidance on radiation protection, the EPA has various statutory authorities and responsibilities regarding regulation of exposure to radiation. The standards and the regulations that EPA has promulgated and proposed with respect to controlling radiation exposures are summarized here.

The U.S. Atomic Energy Act of 1954, as amended, and Reorganization Plan No. 3 granted EPA the authority to establish generally applicable environmental standards for exposure to radionuclides. Pursuant to this authority, in 1977 the EPA issued standards limiting exposure from operations of the light-water reactor nuclear fuel cycle (EPA77b). These standards cover normal operations of the uranium fuel cycle, excluding mining and waste disposal. The standards limit the annual dose equivalent to any member of the public from all phases of the uranium fuel cycle (excluding radon and its daughters) to 25 mrems to the whole body, 75 mrems to the thyroid, and 25 mrems to any other organ. To protect against the buildup of long-lived radionuclides in the environment, the standard also sets normalized emission limits for krypton-85, iodine-129, and plutonium-239 combined with other transuranics with a half-life exceeding one year. The dose limits imposed by the standard cover all exposures resulting from radiation and radionuclide releases to air and water from operations of fuel-cycle facilities.

The development of this standard took into account both the maximum risk to an individual and the overall effect of releases from fuel-cycle operations on the population and balanced these risks against the costs of effluent control in a primarily qualitative way.

Under the authority of the Uranium Mill Tailings Radiation Control Act, the EPA promulgated standards limiting public exposure to radiation and restricting releases of materials from uranium tailings piles (EPA83a). Cleanup standards for land and buildings contaminated with residual radioactive materials from inactive uranium processing sites were also established. In these actions, the Agency sought to balance the radiation risks imposed on individuals and the population in the vicinity of the pile against the feasibility and costs of control.

The Agency first established regulations and criteria for the disposal of radioactive waste into the oceans in 1973 under the authority of the Marine Protection, Research and Sanctuaries Act of 1972. These regulations (40 CFR Parts 220-229), which were revised in 1977, prohibit ocean disposal of high-level radioactive wastes and radiological warfare agents and establish requirements for obtaining ocean disposal permits for other radioactive waste (EPA77a).

In 1982, EPA issued effluent limitations guidelines for the ore mining and dressing point source category under the Clean Water Act. Subpart C - Uranium, Radium and Vanadium Ores Subcategory of 40 CFR Part 440 limits, among other items, the concentrations of radium and uranium in effluent discharges from such mines and prohibits the discharge of process wastewater from uranium mills in dry climates.

Under the authority of the Safe Drinking Water Act, the EPA issued interim regulations covering the permissible levels of radium, gross alpha, manmade beta, and photon-emitting contaminants in community water systems (EPA76). The limits are expressed in picocuries/liter. The limits chosen for manmade beta- and photon-emitters equate to approximately 4 mrems/year whole-body or organ dose to the most exposed individual. In the background information for the standard, the 4 mrems/year exposure through a single pathway that the standard permits is explicitly compared with the overall population standard of 170 mrems/year, and the conclusion is expressed that the roughly 40-fold decrease is appropriate for a single pathway.

Section 122 of the Clean Air Act amendments of 1977 (Public Law 95-95) directed the Administrator of EPA to review all relevant information and determine if emissions of hazardous pollutants into air will cause or contribute to air pollution that may reasonably be expected to endanger public health. In December 1979, EPA designated radionuclides as hazardous air pollutants under Section 112 of the Act. On February 6, 1985, and April 17, 1985, EPA published National Emission Standards for radionuclides for selected sources (EPA85a, 85b).

In 1982, under the authority of the U.S. Atomic Energy Act of 1954, as amended, the EPA proposed standards for disposal of spent fuel, high-level wastes, and transuranic elements (EPA82). The proposed standards establish two different limits: (1) during the active waste-disposal phase, operations at the repository must be conducted so that no member of the public receives a dose greater than that allowed for other phases of the uranium fuel cycle; and (2) once the repository is closed, exposure is to be controlled by limiting releases. The release limits were derived by summing, over long time periods, the estimated risks to all persons exposed to radioactive materials released into the environment. The uncertainties involved in estimating the performance of a theoretical repository led to this unusual approach, and the proposed standards admonish the agencies responsible for constructing and operating such repositories to take steps to reduce releases below the upper bounds given in the standards to the extent reasonably achievable.

2.5 Nuclear Regulatory Commission

Under the authority of the Atomic Energy Act of 1954, as amended, the U.S. Nuclear Regulatory Commission (NRC) is responsible for licensing and regulating the use of byproduct, source, and special nuclear material, and for assuring that all licensed activities are conducted in a manner that protects public health and safety. The Federal guidance on radiation protection applies directly to the NRC; therefore, the NRC must assure that none of the operations of its licensees exposes an individual of the public to more than 0.5 rem/year from all pathways. The dose limits imposed by the EPA's standard for uranium fuel-cycle facilities (40 CFR Part 190) also apply to the fuel-cycle facilities licensed by the NRC. These facilities are prohibited from releasing radioactive effluents in amounts that would result in doses greater than the 25 mrems/year limit imposed by that standard.

Also NRC facilities are required to operate in accordance with the requirements of the Clean Air Act (40 CFR Part 61), which limits radionuclide emissions to air to that amount which will cause a dose equivalent of 25 mrems/year to the whole body or 75 mrems/year to the critical organ of any member of the public.

The NRC exercises its statutory authority by imposing a combination of design criteria, operating parameters, and license conditions at the time of construction and licensing. It assures that the license conditions are fulfilled through inspection and enforcement. The NRC licenses more than 7000 users of radioactivity.

2.5.1 Fuel Cycle Licenses

The NRC does not use the term "fuel cycle facilities" to define its classes of licensees. The term is used here to coincide with the EPA use of the term in its standard for uranium fuel cycle facilities. As a practical matter, this term includes the NRC's large source and special nuclear material, and production and utilization facilities. The NRC's regulations require an analysis of probable radioactive effluents and their effects on the population near fuel cycle facilities. The NRC also assures that all exposures are as low as reasonably achievable by imposing design criteria and specific equipment requirements on the licensees. After a license has been issued, fuel-cycle licensees must monitor their emissions and take environmental measurements to assure that the design criteria and license conditions have been met. For practical purposes, the NRC adopted the maximum permissible concentrations developed by the NCRP to relate effluent concentrations to exposure.

In the 1970's, the NRC formalized the implementation of as low as reasonably achievable exposure levels by issuing a regulatory guide for as low as reasonably achievable design criteria. This coincided with a decision to adopt, as a design criterion, a maximum annual permissible dose of 5 mrems from a single nuclear electric generating station. The 5-mrem limit applies to the most exposed individual actually living in the vicinity of the reactor, and refers to whole-body doses from external radiation by the air pathway (NRC77).

2.5.2 Radioactive Waste Disposal Licenses

The NRC's requirements for radioactive waste disposal are contained in 10 CFR Part 60, Disposal of High-Level Radioactive Wastes in Geologic Repositories: Technical Criteria (NRC83); 10 CFR Parts 2, 19, 20, 21, 30, 40, 51, 60, and 70, Disposal of High-Level Radioactive Wastes in Geologic Repositories: Licensing Procedures (NRC81); 10 CFR Part 61, Licensing Requirements for Land Disposal of Radioactive Waste (NRC82); and 10 CFR Part 40, Uranium Mill Licensing Requirements (NRC80). NRC is expected to make certain revisions to 10 CFR Part 60 to bring them into full consistency with the 40 CFR Part 191 issued by EPA.

2.6 Department of Energy

The U.S. Department of Energy (DOE) operates a complex of national laboratories and weapons facilities. These facilities are not licensed by the NRC. Under the U.S. Atomic Energy Act of 1954, as amended, the DOE is responsible for keeping radionuclide emissions at these facilities as low as reasonably achievable (ALARA). The EPA has promulgated a final standard, consistent with the requirements of the Clean Air Act, that limits radionuclide air emissions from DOE facilities to that amount which will cause a dose equivalent of 25 mrems/year to the whole body or 75 mrems/year to the critical organ of any member of the public. These limits generally reflect current emission levels achieved by existing control technology and operating practices at DOE facilities (EPA85a).

For practical purposes, the DOE has adopted the NCRP's maximum permissible concentrations in air and water as a workable way to assure that the annual dose limits of 0.5 rem whole-body and 1.5 rems to any organ are being observed. The DOE also has a requirement that all doses be kept as low as is reasonably achievable, but the contractors that operate the various DOE sites have a great deal of latitude in implementing policies and procedures to assure that all doses are kept to the lowest possible level.

The DOE assures that its operations are within its operating guidelines by requiring its contractors to maintain radiation monitoring systems around each of its sites and to report the results in an annual summary report. New facilities and modifications to existing facilities are subject to extensive design criteria reviews and require the preparation of environmental impact statements pursuant to the National Environmental Policy Act of 1970 (NEPA70). Since the mid-1970's, the DOE initiated a systematic effluent-reduction program that resulted in the upgrading of many facilities and effected a corresponding reduction in the effluents (including airborne and liquid radioactive materials) released to the environment.

The DOE has developed and issued general guidelines in 10 CFR Part 960 (DOE84) for the recommendation of sites for the disposal of high-level radioactive waste and spent nuclear fuel in geologic formations. The guidelines are to be used in various steps of the site selection process and are to be compatible with the regulations issued by the NRC in 10 CFR Part 60 and by the standards issued by the EPA in 40 CFR Part 191. These guidelines establish performance objectives for a geologic repository system, define the basic technical requirements that candidate sites must meet, and specify how the DOE will implement the site-selection process.

2.7 Department of Transportation

The U.S. Department of Transportation (DOT) has statutory responsibility for regulating the shipment and transportation of radioactive materials. This authority includes the responsibility to protect the public from exposure to radioactive materials while they are in transit. For practical purposes, the DOT has implemented its authority through the specification of performance standards for shipment containers, and by setting maximum exposure rates from any package containing radioactive materials. These limits were set to assure compliance with the Federal guidance for occupational exposure, and they are believed to be sufficient to protect the public from exposure. The DOT also controls potential public exposure by managing the routing of radioactive shipments to avoid densely populated areas.

2.8 State Agencies

States have important authority for protecting the public from the hazards associated with ionizing radiation. Twenty-six States have assumed NRC's inspection, enforcement, and licensing responsibilities for users of source and byproduct materials and users of small quantities of special nuclear material. These "NRC-agreement States," which license and regulate more than 11,500 users of radiation and radioactive materials, are bound by formal agreements to adopt requirements consistent with those imposed by the NRC. The NRC continues to perform this function for all licensable uses of source, byproduct, and special nuclear material in the 24 States that are not agreement States.

State and public participation in the planning and development of high-level waste repositories is essential in order to promote public confidence in the safety of disposal of these wastes. States which are identified by the Secretary of Energy as having one or more acceptable sites for a high-level waste repository may disapprove the site designation and submit to the Congress a notice of disapproval (NWPA83). This notice must be accompanied by a statement of reasons explaining why the recommended repository site has been disapproved.

Grants are available to States with acceptable sites so that a State may: 1) determine potential economic, social, public health and safety, and environmental impacts of the repository on the State and its residents; 2) develop a request for impact assistance; 3) engage in any monitoring, testing, or evaluation activities with respect to site characterization programs; 4) provide information to its residences with respect to the site; and 5) request information from, and make comments and recommendations to, the Secretary of Energy with respect to the site (NWPA83).

After construction of a high-level waste repository is authorized, financial and technical assistance will be provided to the State by the Secretary of Energy to mitigate the impact of the development of the repository on the State. The State must provide a report on economic, social, public health and safety, and environmental impacts that are likely as a result of the development of a repository at the specified site. The State will be notified of the transportation of any high-level radioactive waste or spent fuel that is brought into the State for disposal at the repository site and can conduct reasonable independent monitoring and testing of activities on the repository site (NWPA83).

2.9 Indian Tribes

If a recommended high-level radioactive waste repository site is located on the reservation of an Indian tribe, the tribe may disapprove the site designation and submit to Congress a notice of disapproval. As with the State, grants are available to affected Indian tribes so that they may: 1) determine any potential economic, social, public health and safety, and environmental impacts of the repository on the reservation and its residents; 2) develop a request for impact assistance; 3) engage in any monitoring, testing, or evaluation activities with respect to site characterization programs; 4) provide information to the residents of the reservation with respect to the site; and 5) request information from, and make comments and recommendations to, the Secretary of Energy with respect to the site (NWPA83).

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Chapter 3: QUANTITIES, SOURCES, AND CHARACTERISTICS OF SPENT NUCLEAR FUEL AND HIGH-LEVEL AND TRANSURANIC WASTES

3.1 Introduction

Presented in this chapter are current inventories of commercial spent fuels, commercial and U.S. Department of Energy (DOE) high-level radioactive wastes, and DOE transuranic wastes. These inventories were compiled from the most reliable government information. Estimates of generated wastes and spent fuel to the year 2000, based on the latest DOE information and projected U.S. commercial power growth, are also presented. The spent fuel and wastes are characterized according to their volumes (or masses) and their nuclear, physical, and chemical properties.

The radioactive wastes and spent nuclear fuel originate from the commercial nuclear fuel cycle and DOE defense-related activities. The wastes are broadly characterized as high-level waste (HLW) and transuranic (TRU) waste. In addition, an inventory of commercial reactor spent fuel also may require an expansion of current storage or the construction of additional facilities for interim storage, pending the availability of commercial reprocessing facilities, permanent disposal facilities, or monitored retrievable storage.

Both spent fuel and high-level radioactive wastes from reprocessing are intensely radioactive and generate substantial quantities of heat. The radioactivity and heat production continue for long periods of time because the wastes contain a number of long-lived radionuclides. The transuranium elements in particular have long radiological half-lives, generate very little heat, and present a possible hazard to people for tens of thousands of years.

3.2 Spent Nuclear Fuel (EPA82, DOE84a, Bu82, Li79, St79)

In this standard, spent nuclear fuel is defined as fuel that has been withdrawn from a nuclear reactor following irradiation and whose constituent elements have not been separated by reprocessing.

Spent fuel from government, industrial, and commercial sources can be categorized as 1) fuel discharged from commercial light-water reactors (LWR's); 2) fuel elements generated by government-sponsored research and demonstration programs, universities, and industry; 3) fuels from experimental reactors [viz., liquid metal fast breeder reactor (LMFBR) and high-temperature gas-cooled reactors (HTGR)]; 4) U.S. Government-controlled nuclear weapon production reactors; and 5) naval reactor fuels and other Department of Defense (DOD) reactor fuels.

Most (95 percent) of the spent fuels from commercial power reactors are stored at the reactor sites. The rest are stored at the Nuclear Fuel Services (NFS) plant at West Valley, New York, and at the Midwest Fuel Recovery Plant (MFRP) at Morris, Illinois. The NFS plant is now being decommissioned, and the residual fuel quantities stored there are being transferred to other sites. Special fuels are stored at the Savannah River Plant (SRP) in South Carolina and the Idaho Chemical Processing Plant (ICPP) in Idaho. The LMFBR fuel from the Fast Flux Test Facility (FFTF) is stored at Hanford, Washington (HANF), and HTGR spent fuel discharged from the Fort St. Vrain reactor is stored at ICPP. Production and naval reactor fuels are stored at SRP, ICPP, and Hanford, awaiting reprocessing by government-owned facilities.

The fuel currently used in commercial light-water reactors consists of a mixture of uranium-238 and uranium-235 dioxides encased in zirconium alloy (zircaloy) or stainless steel tubes. During reactor operation, fission of the uranium-235 produces energy, neutrons, and fission products. The neutrons produce further fission reactions and thus sustain the chain reaction. The neutrons also convert some of the uranium-238 into plutonium-239, which can fission as uranium-235 can. In time, the fissile uranium-235, which originally constituted some 3 to 4 percent of the enriched fuel, is depleted to such a low level that power production becomes inefficient. Once this occurs, the fuel bundles are deemed "spent" and are removed from the reactor. Typical removal rate is one-third of the fuel, or 30 tons/year per reactor. Reprocessing of commercial spent fuel has been proposed to recover the unfissioned uranium-235 and the plutonium for reuse as a fuel resource, but such reprocessing is not currently taking place.

The radioactive materials in spent fuel fall into two major categories: fission products and actinide elements. Typically, fresh spent fuel contains more than 100 radionuclides as fission products. Fission products of particular importance, because of the quantities produced or their biological hazard, are: strontium-90; technetium-99; iodine-129 and -131; the cesium isotopes 134, 135, and 137; tin-126; and krypton-85 and other noble gas isotopes. The actinides consist of uranium isotopes, transuranic elements (i.e., isotopes with an atomic number greater than 92, including plutonium, curium, americium, and neptunium formed by neutron capture, and their decay products. Spent fuel also contains tritium (hydrogen-3), carbon-14, and other radioactive isotopes created by neutron activation. The exact composition of radionuclides in any given spent-fuel sample depends on the reactor type, the initial fuel composition, the length of time the fuel was irradiated, and the elapsed time since its removal from the reactor core.

3.2.1 Spent Fuel Inventory and Projection (DOE84a)

As of December 31, 1983, there were 10,140 metric tons (t) of spent fuel in inventory from commercial reactor operation. Of this amount, 159 t are stored at the NFS facility and 322.5 t are stored at the MFRP. The remainder is at the reactor sites. The oldest light-water-reactor spent fuel in inventory was discharged in 1970. The historical and projected buildup of the spent fuel inventory and accumulated radioactivity are given in Table 3.2-1. These values do not include the relatively small amount of spent fuel reprocessed by the NFS facility.

Table 3.2-1. Historical and projected mass and radioactivity of commercial spent fuel (DOE84a)

End of calendar year	Mass accumulated (t)	Radioactivity accumulated (10^6 Ci)
1970	28	134
1975	1,449	4,057
1980	6,496	10,236
1983	10,140	12,879
1985	12,449	13,178
1990	21,121	23,176
1995	31,559	29,456
2000	42,812	35,674

The activity of spent fuel depends primarily on its age. As the spent fuel ages, many of the short-lived fission products decay. Calculations of waste activities 10 years after removal from the reactor, with consideration being given only to radionuclides (fission products and heavy elements) with half-lives greater than 20 years, show that the 1983 activity of the 10,400 t of spent fuel corresponds to about 1.6 billion curies.

The projected inventory of spent fuel (Table 3.2-1) was based on the projected installed nuclear capacities given in Table 3.2-2.

Table 3.2-2. Historical and projected installed nuclear electric power capacity (DOE84a)

End of calendar year	Total GW(e)	End of calendar year	Total GW(e)
1960	0.2	1983	59.3
1965	0.8	1985	83.5
1970	4.7	1990	109.6
1975	34.9	2000	121.5
1980	49.8		

The spent fuel from special research and test reactors is shipped to either the SRP or the ICPP for indefinite storage or eventual reprocessing. The production and naval reactor fuels are stored at SRP, ICPP, and Hanford for routine reprocessing. As of December 31, 1983, the special spent fuel inventory was over 4500 Kg U-235, whereas the special Fort St. Vrain HTGR fuel in storage was 2.5 metric tons.

3.3 High-level Radioactive Wastes (EPA82, DOE84a, Li79, St79, DOE80)

The standard defines high-level radioactive wastes as the highly radioactive materials resulting from the reprocessing of spent nuclear fuel, including liquid waste produced directly in reprocessing and any solid material derived from such liquid waste. This definition is the same as listed in the NWPA; however, it is slightly different from the previous EPA definition in the ocean dumping regulations, 40 CFR Parts 220-227, and the NRC definition in 10 CFR Part 60. Federal regulations require that commercial high-level waste generated in the future be converted to a solid within 5 years.

The fission products, actinides, and neutron-activated products of particular importance are the same for HLW as those listed for the spent fuel assemblies.

Weapons program reactors are operated (by DOE contractors) to produce plutonium. Reprocessing to recover the plutonium is an integral part of the weapons program operations. Naval propulsion reactor fuel elements are also reprocessed to recover the highly enriched uranium they contain.

High-level radioactive waste that is generated by the reprocessing of spent reactor fuel and targets contains more than 99 percent of the nonvolatile fission products produced in the fuel or targets during reactor operation. It generally contains about 0.5 percent of the uranium and plutonium in the original fuel. Most of the current HLW inventory, which is the result of DOE national defense activities, is stored at the Savannah River Plant, the ICPP at the Idaho National Engineering Laboratory (INEL), and the Hanford sites. A small amount of commercial HLW was generated at the Nuclear Fuel Services Plant at West Valley, New York, from 1966 to 1972. These wastes have been through one or more treatment steps (i.e, neutralization, precipitation, decantation, evaporation, etc.). Their volumes depend greatly on the steps they have been through. They must be incorporated into a stable solid medium (e.g., glass) for final disposal, and the volumes of these interim wastes will be greatly reduced once this has been accomplished.

The DOE/defense HLW at INEL results from reprocessing nuclear fuels from naval propulsion reactors and special research and test reactors. The bulk of this waste, which is acidic, has been converted to a stable, granular solid (calcine). At SRP and HANF, the acidic waste from reprocessing defense reactor fuel is or has been made alkaline by the addition of a caustic and stored in tanks. During storage, these alkaline wastes separate into three or four phases: liquid, sludge, slurry, and salt cake. The relative proportions of liquid and salt cake depend on how much water is removed by waste evaporators during waste management operations. The condensed water may be recycled within the facility or decontaminated further and discharged.

The commercial HLW at West Valley consists of both alkaline and acidic waste. The alkaline waste was generated by reprocessing commercial power reactor fuels and some Hanford N-Reactor fuels, whereas the acidic waste was generated by reprocessing a small amount of commercial fuel containing thorium.

The inventories of HLW in storage at the end of 1983 are listed in Table 3.3-1 (by volume) and Table 3.3-2 (by radioactivity). Projected volume and radioactivity data for DOE/defense, West Valley, and future commercial HLW are given in Table 3.3-3.

3.3.1 HLW Inventories at SRP

The approximately 111,000 m^3 of alkaline HLW that has accumulated at the SRP over the past three decades is stored in high-integrity, double-walled, carbon-steel tanks. The current inventories (Tables 3.3-1 and 3.3-2) consist of alkaline liquid, sludge, and salt cake that were generated primarily by the PUREX reprocessing of nuclear fuels and targets from plutonium production reactors. As generated, most of the waste is acid, and the sludge is formed after treatment with caustic and after aging. Salt cake results when the supernatant liquor is concentrated in evaporators.

3.3.2 HLW Inventories at INEL

The 9700 m^3 of HLW stored at INEL is at the Idaho Chemical Processing Plant; it consists of 6900 m^3 of liquid waste and 2800 m^3 of calcine (Tables 3.3-1 and 3.3-2). Liquid HLW is generated at ICPP primarily by the reprocessing of spent fuel from the national defense (naval propulsion nuclear reactors) and reactor testing programs; a small amount is generated by reprocessing fuel from nondefense research reactors. This acidic waste is stored in large, doubly contained, underground, stainless steel tanks. The waste is then converted to a calcine, after which it is stored in stainless steel bins housed in reinforced concrete vaults.

3.3.3 HLW Inventories at HANF

The 203,000 m^3 of alkaline HLW stored at HANF is in four phases: liquid, sludge, slurry, and salt cake. This waste, which has been accumulating since 1944, was generated by reprocessing production reactor fuel for the recovery of plutonium, uranium, and neptunium for defense and other Federal programs. Reprocessing was suspended from 1972 until November 1983. Most of the high-heat-emitting isotopes (^{90}Sr and ^{137}Cs, plus their daughters) have been removed from the old waste, converted to solids as strontium fluoride and cesium chloride, placed in double-walled capsules, and stored in water basins. The liquid, sludge, slurry, and salt cake wastes (Tables 3.3-1 and 3.3-2) are stored in underground concrete tanks with carbon steel liners.

Table 3.3-1. Current volume of HLW in storage by site through 1983 (DOE84a)

Site	Volume (10^3 m^3)						
	Liquid	Sludge	Salt cake	Slurry	Calcine	Capsules[(a)]	Total
Defense							
Savannah River Plant	65.9	12.8	32.7	(b)	(b)	(b)	111.4
Idaho Chemical Processing Plant	6.9	(b)	(b)	(b)	2.8	(b)	9.7
Hanford	57.0	47.0	95.0	4.0	(b)	0.0049	203.0
Subtotal	129.8	59.8	127.7	4.0	2.8	0.0049	324.1
Commercial							
Nuclear Fuel Services							
Acid waste	0.045	(b)	(b)	(b)	(b)	(b)	0.045
Alkaline waste	2.1	0.17	(b)	(b)	(b)	(b)	2.27
Subtotal	2.145	0.17	(b)	(b)	(b)	(b)	2.315
Grand total	131.9	60.0	127.7	4.0	2.8	0.0049	326.4

(a) Capsules contain either strontium (^{90}Sr-^{90}Y) fluoride or cesium (^{137}Cs-^{137m}Ba) chloride.

(b) Not applicable.

Table 3.3-2. Current radioactivity of HLW in storage by site through 1983 (DOE84a)

Site	Radioactivity[a] (10^6 Ci)						
	Liquid	Sludge	Salt cake	Slurry	Calcine	Capsules	Total
Defense							
Savannah River Plant	85.9	509.2	181.1	(b)	(b)	(b)	776.2
Idaho Chemical Processing Plant	16.2	(b)	(b)	(b)	48.6	(b)	64.8
Hanford	33.0	143.3	14.4	0.22	(b)	283.3[c]	474.2
Subtotal	135.1	652.5	195.5	0.22	48.6	283.3	1315.2
Commercial							
Nuclear Fuel Services							
Acid waste	2.95	(b)	(b)	(b)	(b)	(b)	2.95
Alkaline waste	15.2	16.7	(b)	(b)	(b)	(b)	31.9
Subtotal	18.15	16.7	(b)	(b)	(b)	(b)	34.85
Grand total	153.2	669.2	195.5	0.22	48.6	283.3	1350.0

[a] Calculated values allowing for radioactive decay.

[b] Not applicable.

[c] Includes strontium and cesium in capsules and separated concentrates that are awaiting encapsulation. The quantity of $^{90}Sr-^{90}Y$ is 1.074 x 10^8 Ci and that of $^{137}Cs-^{137m}Ba$ is 1.759 x 10^8 Ci.

Table 3.3-3. Historical and projected volume and associated radioactivity of HLW in storage by site through 2000 (DOE84a)

End of calendar year	Volume (10^3 m^3)								Radioactivity (10^6 Ci)
	Liquid	Sludge	Salt cake	Slurry	Calcine	Capsules[(a)]	Glass[(b)]	Total	Total
					Savannah River Plant				
1980	59.8	10.5	26.4	--	--	--	--	96.7	699.0
1983	65.9	12.8	32.7	--	--	--	--	111.4	776.2
1985	55.4	14.0	41.2	--	--	--	--	110.6	813.1
1990	51.4	15.0	50.3	--	--	--	0.3	117.0	790.6
1995	37.6	14.3	44.3	--	--	--	2.0	98.2	751.0
2000	29.0	13.5	36.6	--	--	--	3.6	82.7	698.9
					Idaho Chemical Processing Plant				
1980	9.34	--	--	--	2.07	--	--	11.4	53.4
1983	6.9	--	--	--	2.8	--	--	9.7	64.8
1985	7.0	--	--	--	3.0	--	--	10.0	73.8
1990	5.2	--	--	--	4.9	--	--	10.1	90.3
1995	5.7	--	--	--	6.8	--	--	12.5	140.7
2000	2.9	--	--	--	11.0	--	--	13.9	240.8
					Hanford				
1980	39.0	49.0	95.0	(c)	--	0.0017	--	183.0	557.6
1983	57.0	47.0	95.0	4.0	--	0.0049	--	203.0	474.2
1985	62.0	50.0	95.0	6.0	--	0.0049	--	213.0	560.5
1990	57.0	55.0	95.0	8.0	--	0.0103	--	215.0	664.6
1995	57.0	56.0	95.0	9.0	--	0.0105	--	217.0	574.4
2000	57.0	56.0	95.0	9.0	--	0.0105	--	217.0	430.4

(continued)

Table 3.3-3. Historical and projected volume and associated radioactivity of HLW in storage by site through 2000 (DOE84a) (continued)

End of calendar year	Volume (10^3 m^3)								Radioactivity (10^6 Ci)
	Liquid	Sludge	Salt cake	Slurry	Calcine	Capsules[(a)]	Glass[(b)]	Total	Total
					Nuclear Fuel Services				
1980	2.15	0.047	--	--	--	--	--	2.2	37.7
1983	2.145	0.170	--	--	--	--	--	2.315	103.5
1985	2.145	0.170	--	--	--	--	--	2.315	98.0
1990	--	--	--	--	--	--	0.159	0.159	86.6
1995	--	--	--	--	--	--	0.159	0.159	76.5
2000	--	--	--	--	--	--	0.159	0.159	67.5

(a) Includes strontium and cesium in capsules and separated concentrates that are to be encapsulated.

(b) Glass may be in storage at the site, in transit to a repository, or in a repository.

(c) Slurry included with sludge.

3.3.4 HLW Inventories at NFS

The 2315 m^3 of HLW stored at NFS consists of 2270 m^3 of alkaline waste and only 45 m^3 of acid waste. The alkaline waste was generated by reprocessing commercial and some Hanford N-Reactor spent fuels. Initially, all of the waste was highly acid; treatment with excess sodium hydroxide led to the formation of an alkaline sludge. The acid waste now in storage was generated by reprocessing a small batch of thorium-uranium fuel from the Indian Point-1 Reactor. The alkaline waste is stored in an underground carbon-steel tank, and the acid waste is stored in an underground stainless steel tank. Reprocessing at the NFS plant was discontinued in 1972, and no additional HLW has been generated since then. The current inventories of HLW at NFS are presented in Tables 3.3-1 and 3.3-2.

3.3.5 Waste Characterization

A generic characterization of HLW at any site is difficult because the wastes have been generated by several different processes, and several methods have been used to condition the wastes for storage (e.g., evaporation and precipitation). In some instances, several different wastes have been blended. Nonetheless, representative chemical and radionuclide compositions for HLW at SRP, ICPP, HANF, and NFS can be found in some sources (DOE84a, Li79, DOE80).

As with spent fuel, HLW radioactivity levels depend on age. To bring the activity into perspective, calculations showed that fission products and heavy element radionuclides with half-lives exceeding 20 years in the existing HLW are estimated to be about 700 million curies.

3.3.6 Projections

Projections for HLW (volume and radioactivity) by source are presented in Table 3.3-3. The projections for SRP are based on the restarting of the L-Reactor (fall of 1985) and initial operation of the Defense Waste Processing Facility (DWPF) in late 1989, with the first radioactive glass to be made in 1990.

The ICPP projections are based on predicted fuel deliveries and estimates of fuel reprocessing and waste management operations. The HANF projections are based on the shutdown of the N-Reactor in 1983 and the restarting of the fuel reprocessing plant in November 1983, with operation projected to continue through 1993. The HLW at ICPP and HANF are not incorporated in glass because such processes are not yet available there. At NFS, vitrification of the waste is scheduled to begin in mid-1988 and to be completed by the end of 1989.

3.4 Transuranic Wastes (DOE84a, Li79, DOE80, Ja83, Br81, DOE84b)

The standard defines transuranic wastes as wastes containing more than 100 nanocuries of alpha-emitting transuranic isotopes, with half-lives greater than 20 years, per gram of waste. TRU waste was originally defined by DOE as "...solid material that is contaminated to greater than 10 nCi/g with certain alpha-emitting radionuclides of long half-life and highly specific radiotoxicity." However, this definition was recently revised by DOE to read that "TRU waste is material having no significant economic value which, at the end of institutional control periods, is contaminated with alpha-emitting radionuclides with atomic numbers greater than 92 and half-lives greater than 20 years, in concentrations greater than 100 nCi/g" (DOE84b). Alpha-emitting transuranic nuclides represent a special type of hazard because of their long half-lives and high radiotoxicity.

Most of the nuclides that make up TRU wastes have very long half-lives and low specific activities. Although a few daughter products have energetic gamma emissions, their most significant hazard is due to alpha radiation emissions. Most TRU wastes can be handled with just the shielding that is provided by the waste package itself. These wastes are classified as "contact-handled" TRU wastes. A smaller volume may be contaminated with sufficient beta, gamma, or neutron activity to require remote handling. Also, heat generation in stored TRU waste is not a factor affecting how closely packages can be stored; however, avoiding the production of a critical mass as a result of densely-stored material must always be considered.

Most TRU wastes are generated in DOE defense-related activities at the Rocky Flats Plant (RFP), Hanford Facilities, and the Los Alamos National Laboratory (LANL). Nearly one-half of all TRU waste comes from weapons components manufactured at RFP and subsequent plutonium recovery at these three sites. Smaller amounts are generated at the Oak Ridge National Laboratory (ORNL), SRP, INEL, Argonne National Laboratory (ANL), Mound Facility, Bettis Atomic Power Laboratory, Lawrence Livermore Laboratory, and Battelle-Columbus Laboratory. The second largest source of TRU waste is decontamination and decommissioning (D&D) projects, which account for one-fourth of the total. About one-fifth of TRU wastes come from laboratory activities, which can produce exotic TRU isotopes.

The amounts of TRU wastes from fuel cycle activities are quite small because of the current moratorium on reprocessing and plutonium recycle. The Nuclear Fuel Services' reprocessing of nuclear fuel at West Valley, New York, produced some TRU waste that was disposed of at that site. A small amount of TRU waste is also being generated in industrial and government-sponsored fuel fabrication and research.

3.4.1 Inventories and Characterization

As opposed to other radioactive wastes, TRU wastes represent a group of liquid and solid materials with widely varying chemical and physical properties. These wastes are categorized as contact-handled (CH), i.e.,

Table 3.4-1. Inventories and characteristics of DOE/defense TRU wastes buried through 1983 (DOE84a)

	Values reported by burial site as of Dec. 31, 1983		
Burial site	Volume (m^3)	Mass of TRU elements (kg)	Alpha radioactivity (Ci)
HANF	92,100	350	29,230
INEL	57,100	357	73,267
LANL	11,486	14	6,580
ORNL	6,200	5.6	272
SAND	3	<<1	<<1
SRP	4,520	9.4	54,284
Total	171,409	736	163,633

Table 3.4-2. Inventories and characteristics of DOE/defense waste in TRU retrievable storage through 1983 (DOE84a)

Storage site	Volume (m^3)	Mass of TRU elements (kg)	Alpha radioactivity (Ci)
	Values reported by storage site as of Dec. 31, 1983		
Contact handled[(a)]			
HANF	12,808	340	27,680
INEL	50,958	524.4	171,157
LANL	6,294.8	247.5	138,017
NTS	319.7	7.775	1,318.7
ORNL	450	12.33	21,414
SRP	3,399	98.5	580,761
Subtotal	74,229.5	1,230.5	940,348
Remotely handled[(a)]			
HANF	21.8	5.4	750
INEL	50.69	0.319	24.1
LANL	26.6	1.27	78
ORNL	653	0.613	428
Subtotal	752.09	7.602	1,280.1
Total	74,981.6	1,238.1	941,628

(a) Beginning with 1983, TRU waste inventories are estimated on the basis of DOE Order 5820.2, which defines TRU waste as 100 nCi/g. Prior inventories were estimated on the basis of the earlier definition of TRU waste (10 nCi/g); hence a portion of the volume might be reclassified as non-TRU waste.

Table 3.4-3. Estimated inventories of items that might require special handling and/or treatment as TRU waste (DOE84a)

DOE Burial site	Volume (m^3) of contaminated soil		Mass (kg) of TRU elements in contaminated soil		Alpha radioactivity (Ci) of contaminated soil	
	Solid waste burial	Liquid disposal/spills	Solid waste burial	Liquid disposal/spills	Solid waste burial	Liquid disposal/spills
HANF	27,900	32,000	350	190	30,000	15,900
INEL	56,640-156,000	0	Unknown	0	Unknown	0
LANL	1,000	140	Unknown[a]	0.12	Unknown[a]	8.6
ORNL	12,000-60,000	1,000	Unknown	0.3	Unknown	8
SRP	Up to 38,000	Not reported	9.4	Not reported	54,284	Not reported
Total	135,540-282,900	33,340	359.4	190.42	84,284	15,916.6

(a) The mass of TRU elements and the radioactivity are included in the total inventory of buried waste (see Table 3.4-1). There is no known method of estimating these values for the contaminated soil.

Table 3.4-4. Physical composition of TRU wastes at DOE/defense sites (DOE84a)

	Waste composition (vol. %)		
	Retrievably stored waste		
Waste type	Contact handled	Remotely handled	Buried waste
	HANF		
Absorbed liquids or sludges	5.8		8
Combustibles	23.3	65.1	20
Concreted or cemented sludge	2.4	6.1	5
Filters or filter media	0.5		1
Glass	3.6		8
Metal	48.7	28.8	40
Other	15.7		18
	INEL		
Absorbed liquids or sludges	14.13		(a)
Alpha hot cell waste		80.09	
Combustibles	20.67	4.333	
Concreted or cemented sludges	3.034		
Dirt, gravel, or asphalt	1.802		
Filters or filter media	7.522	11.59	
Glass	2.051		
Laboratory waste		3.513	
Metal	28.02		
Solidified fuel		0.4684	
Other	12.32		
Unknown	10.49		
	LANL		
Absorbed liquids or sludges	18.1		24
Combustibles	16.6		
Concreted or cemented sludges	11.9		44
Dirt, gravel, or asphalt	2.1		
Filters or filter media	2.6		
Glass	0.5		
Metal	39.3		
Other	8.9	100	32
	NTS		
Combustibles	45		
Metal	55		
	ORNL		
Combustibles	56	39	(a)
Filters or filter media	0	2	
Metal	16	40	
Other	28	19	
	SRP		
Combustibles	70		90
Noncombustible	30		10

(a) Data not available to determine composition of buried waste.

Table 3.4-5. Estimated isotopic composition of buried, retrievably stored, and future TRU waste (DOE84a)

	Isotopic composition (wt. %)		
	Retrievably stored waste		
Isotope	Contact handled	Remotely handled	Buried waste
		HANF	
^{238}Pu	0.01	0.05	0.01
^{239}Pu	93.89	86.36	93.89
^{240}Pu	5.75	11.75	5.75
^{241}Pu	0.34	1.63	0.34
^{242}Pu	0.02	0.21	0.02
		INEL	
^{233}U	18.22		
^{238}Pu	0.728	0.05417	0.0084
^{239}Pu	72.36	92.86	90.42
^{240}Pu	4.552	7.001	5.652
^{242}Pu	0.0238	0.0860	0.01554
^{241}Am	4.119		3.903
		LANL	
^{233}U	1.7		20.0
^{236}Pu	2.8E-06		1.0E-08
^{238}Pu	2.25	0.014	0.017
^{239}Pu	87.92	93.55	62.68
^{240}Pu	5.59	5.89	3.94
^{241}Pu	0.518	0.536	0.359
^{242}Pu	0.524	0.023	0.015
^{241}Am	1.4		13.0
^{226}Ra	<0.1		
^{237}Np	<0.1		
^{244}Cm	<0.1		
		NTS	
^{237}Np	9.96		
^{238}Pu	0.013		
^{239}Pu	83.54		
^{240}Pu	5.26		
^{241}Pu	0.479		
^{242}Pu	0.021		
^{241}Am	0.631		
^{244}Cm	0.112		

(continued)

Table 3.4-5. Estimated isotopic composition of buried, retrievably stored, and future TRU waste (DOE84a) (continued)

	Isotopic composition (wt. %)		
	Retrievably stored waste		
Isotope	Contact handled	Remotely handled	Buried waste
	ORNL		
^{233}U	63.5	38.6	Unknown
^{237}Np	3.1	0	"
^{238}Pu	3.9	trace	"
^{239}Pu	22.8	59.8	"
^{240}Pu	4.0	0	"
^{241}Pu	0.3	0	"
^{242}Pu	0.2	0	"
^{241}Am	1.3	1.1	"
^{243}Am	0.4	trace	"
^{242}Cm	trace	0	"
^{244}Cm	0.5	0.5	"
^{246}Cm	trace	0	"
^{252}Cf	trace	trace	"
^{249}Bk	trace	trace	"
	SRP(a) (^{239}Pu operations)		
^{238}Pu	0.02		0.02
^{239}Pu	93-94		93-94
^{240}Pu	6		6
^{241}Pu	0.5		0.5
^{242}Pu	0.07		0.07
	SRP(a) (^{238}Pu operations)		
^{238}Pu	80-83		80-83
^{239}Pu	17-20		17-20
^{240}Pu	2		2
^{241}Pu	0.4		0.4
^{242}Pu	0.1		0.1

(a) SRP estimates that 50 percent of the contact-handled waste is from ^{239}Pu operations and 50 percent from ^{238}Pu operations.

Table 3.4-6. Current inventories and projections of DOE buried and stored TRU waste from defense activities (DOE84a)

End of calendar year	Volume (10^3 m^3) Accumulation	Radioactivity (10^6 Ci) Accumulation	Mass (kg) Accumulation
	Buried(a)		
1983	171.4	0.3	736.0
1985	171.4	0.3	735.9
1990	171.4	0.3	735.9
1995	171.4	0.2	735.9
2000	171.4	0.2	735.9
	Stored(a),(b)		
1983	75.0	1.5	1238.1
1985	85.5	1.8	1417.9
1990	112.0	2.5	1816.4
1995	140.3	3.3	2369.4
2000	168.7	3.9	2878.8

(a) Beginning with 1983, TRU waste inventories have been estimated on the basis of DOE Order 5820.2, which defines TRU waste as 100 nCi/g. Prior inventories were estimated using the earlier definition of TRU waste (10 nCi/g); hence a portion of the volume might be reclassified as non-TRU waste.

(b) Includes TRU wastes that will be shipped to the Waste Isolation Pilot Plant.

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Chapter 4: PLANNED DISPOSAL PROGRAMS

4.1 Introduction

Since the inception of the nuclear age in the 1940's, the Federal government has assumed ultimate responsibility for the care and disposal of high-level radioactive wastes, regardless of their source, in order to protect the public health and safety and the environment.

The Civilian Radioactive Waste Management (CRWM) Program, formerly called the National Waste Terminal Storage (NWTS) Program, was established in 1976 by DOE's predecessor, the Energy Research and Development Administration (ERDA), to develop technology and provide facilities for the safe, environmentally acceptable, permanent disposal of high-level nuclear waste. Included in the HLW are wastes from both commercial and defense sources, such as spent fuel from nuclear power reactors, accumulations of wastes from production of nuclear weapons, and solidified wastes from fuel reprocessing.

The Federal laws defining DOE's responsibility for the long-term management of HLW specify that the DOE must provide facilities for the successful isolation of HLW from the environment in federally licensed and federally owned repositories for as long as the wastes present a significant hazard (AEA54, ERA74, DEOA74, NWPA82). The Nuclear Waste Policy Act of 1982 (NWPA), enacted January 7, 1983, as Public Law 97-425, confirmed the responsibility of the DOE for management of high-level radioactive waste. The NWPA also confirmed EPA's role in setting general standards and the Nuclear Regulatory Commission's (NRC's) role to act as the licensing agent. The NWPA directed the DOE to provide safe facilities for isolation of high-level radioactive wastes from the environment.

As directed by the NWPA, development work has been performed to define methods for disposal of spent fuel and solidified high-level and transuranic radioactive wastes at the direction of Congress. The development work is being concentrated on mined geological repositories. Such repositories would be constructed in suitable host media at depths greater than 300 meters by conventional mining techniques. Suggested host media include granite, basalt, volcanic tuff, and salt. Wastes in canisters would be placed in holes in the mine floor. When the repository is full, the holes and shafts would be backfilled. After a validation period, during which the wastes could be retrieved, the site would be permanently sealed. Protection would be provided by a stable and insoluble waste form, a durable canister, a stable host medium, and low migration potential for radionuclides through the environment around the host rock. Mined geological repositories are expected to be available for use before any other equally suitable disposal method can be arranged. The NWPA set the disposal capacity of the first repository at 70,000 t of heavy metal.

The Waste Isolation Pilot Plant (WIPP) now under development near Carlsbad, New Mexico, will provide a research and development facility to demonstrate the safe disposal of transuranic radioactive wastes resulting from U.S. defense activities and programs.

4.2 Civilian Radioactive Waste Management Program (DOE82, DOE84a,b)

The CRWM Program emphasizes deep underground disposal in excavated repositories located in geologically stable bodies of rock. Rock types currently being considered include bedded salt deposits, salt domes, basalt, tuff, and crystalline rocks. These rock types are being analyzed at different localities within the co-terminous United States (see Figure 4.2-1) under four projects that are separate from but coordinated with the CRWM Program:

(1) The Salt Repository Project (for bedded salt deposits and salt domes).

(2) The Basalt Waste Isolation Project (for basalt).

(3) The Nevada Nuclear Waste Storage Investigations (for tuff).

(4) The Crystalline Repository Project (for crystalline rocks).

The process for siting the geologic repositories is defined in the NWPA, including a sequence of the steps that form the basis for the strategy to achieve operation of a safe, environmentally sound, licensed geologic repository by 1998.

4.2.1 First Commercial Repository (DOE82, DOE84a-d)

The NWPA requires that the DOE nominate at least five sites to the President and recommend three candidate sites for characterization as possible locations for the first Federal repository. The rock types being considered as potential hosts for the first repository are salt, basalt (a fine-grained rock formed by the solidification of lava), and tuff (compacted volcanic ash). This process is to be repeated for the second repository.

In February, 1983, as required by NWPA, the DOE formally identified the nine potentially acceptable sites being considered for the first repository (see Figure 4.2-2). They include:

- A Nevada site in tuff.

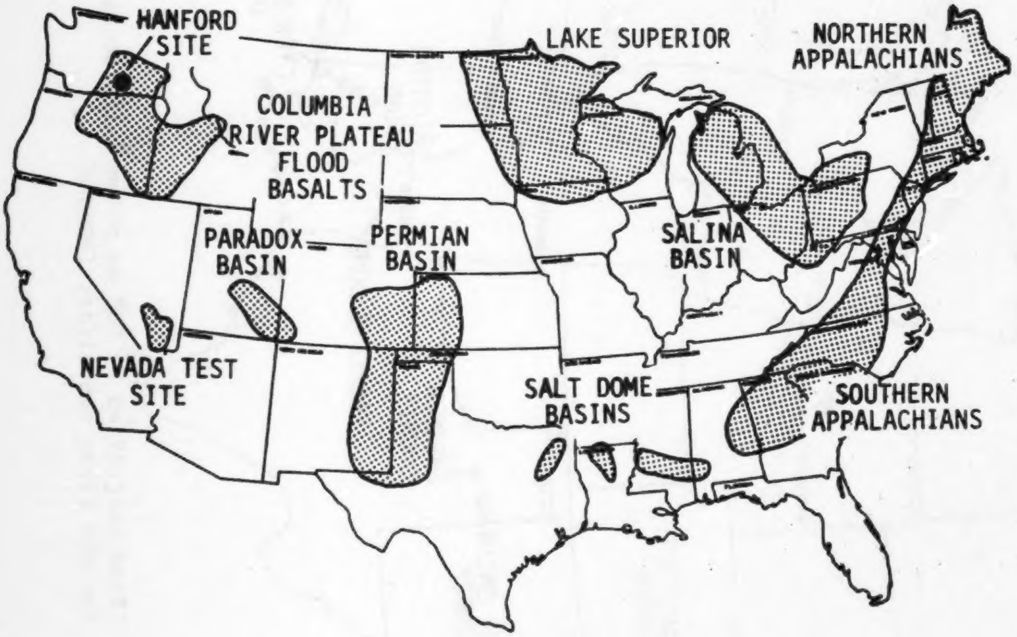

Figure 4.2-1. Regions identified by DOE as under consideration for geological disposal of high-level nuclear waste (DOE82).

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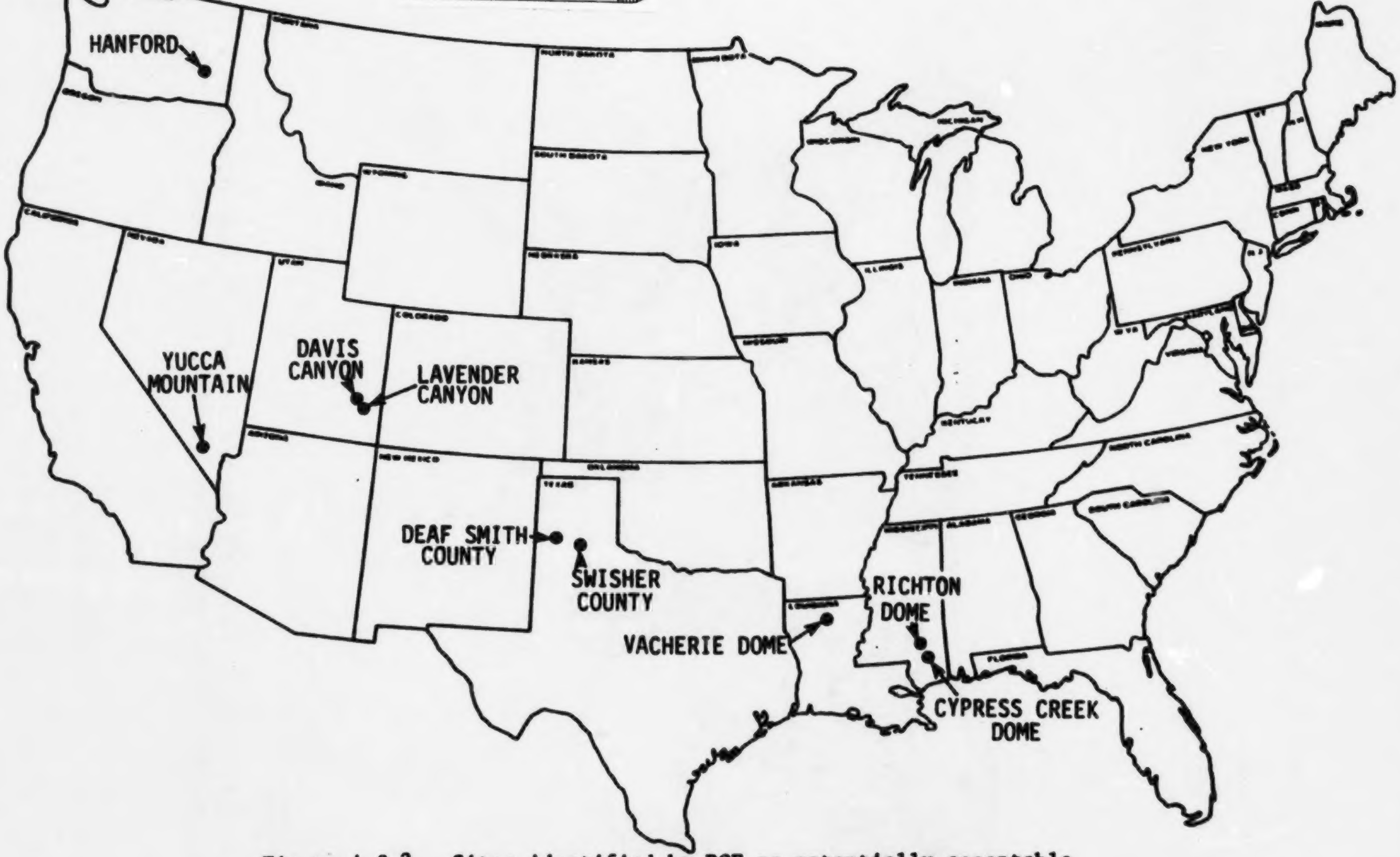

Figure 4.2-2. Sites identified by DOE as potentially acceptable for the first repository (DOE84d).

- A Washington site in basalt.
- Two Texas sites in bedded salt.
- Two Utah sites in bedded salt.
- One Louisiana site in a salt dome.
- Two Mississippi sites in salt domes.

Draft Environmental Assessments have been prepared for all nine potentially acceptable sites (DOE84e-m). In December 1984, five sites were tentatively nominated as being suitable for site characterization: Yucca Mountain in Nevada; Richton Dome in Mississippi; Deaf Smith County in Texas; Davis Canyon in Utah; and Hanford in Washington. Three of these (Yucca Mountain, Deaf Smith County, and Hanford) were recommended by DOE as tentative choices for site characterization.

4.2.2 Second Commercial Repository (NWPA82, DOE82, DOE84b)

In accordance with the NWPA, a separate process of nominations and recommendations will be conducted for a second repository site, which is to be identified by 1990. The NWPA permits sites characterized for the first repository to be nominated for the second repository if not selected as the first site. In addition to crystalline rocks, potential host rocks for the second repository are salt, tuff, and basalt.

As part of its efforts to determine potentially acceptable sites for a second repository, DOE is conducting literature studies on crystalline rock in the following 17 states: Connecticut, Georgia, Maine, Maryland, Massachusetts, Michigan, Minnesota, New Hampshire, New Jersey, New York, North Carolina, Pennsylvania, Rhode Island, South Carolina, Vermont, Virginia, and Wisconsin.

4.3 Geological Media

The characteristics of the various geological media being considered are important in understanding the issues of high-level radioactive waste disposal.

4.3.1 Salt Media (DOE82, DOE83a, DOE84e-g,i-l, Bu82)

Both bedded and domed rock salt are being investigated by DOE's CRWM Program as a suitable host rock for the long-term isolation of high-level radioactive waste. Salt is suitable as a host rock because of its structural strength, radiation-shielding capability, high plasticity (which enables fractures to self-seal at repository depths), low moisture content, and low permeability. In addition, salt deposits are abundant in the United States, and the cost of mining is low. A desirable feature of many bedded salt basins is their relatively simple structure, from which the stratigraphy of a wide area near the repository can be projected. Although salt deposits are widespread, the salt itself and associated deposits of potash or hydrocarbons are resources that could increase the probability of accidental human intrusion into a repository. The solubility of rock salt is two orders of magnitude greater than any other potential host rock and this is important in the analysis of potential failure modes for salt.

4.3.2 Tuffs (DOE82, DOE84h, Bu82, K180)

The two forms of tuff considered for repository use are quite different. The first form is densely welded tuff (i.e., one in which the glass shards became fused because they were hot and plastic when deposited). This form has high density, low porosity and water content, and the capability to withstand the temperatures generated by radioactive waste. The compressive strength, thermal conductivity, and thermal expansion of densely welded tuff are comparable to those of basalt.

The second form of tuff of interest is a zeolitic tuff (i.e., a nonwelded tuff containing zeolite, a hydrous silicate of open molecular structure). This form has low density, high porosity, low interstitial permeability, high water content, extremely high sorptive properties, moderate compressive strength, and moderate thermal conductivity. Dehydration of some zeolites begins at about 100°C, and unless the water released is able to escape through the rock, it could contribute to changes in stress that could result in fracture. An increase in temperature can also cause some zeolites to decompose to new minerals of lower sorptive capacity.

The repository design concept is to place radioactive waste in thermally stable welded tuff, where it would gain a significant benefit from highly sorptive barriers of zeolitic tuff underlying, and where possible, overlying the welded tuff.

Occurrences of welded and zeolitic tuffs are widespread, and some occur in thick sections in the western states; however, their homogeneity and hydrologic properties have not been characterized. Most of these tuffs are relatively young geologically; they have been broken into blocks tens of kilometers in size by tectonic forces that were active during and after the time the tuffs were formed through volcanic eruptions.

4.3.3 Basalt (DOE82, DOE84m, Bu82, K180)

Basalt is the potential host rock at the Hanford Site in Washington, where it occurs in a thick section near the middle of the extensive basalt flows of the Columbia Plateau. Thick basaltic sections also occur in Idaho and Oregon. These basaltic terrains are geologically young, and earthquakes have caused possible surface manifestations; however, no faults are known to jeopardize the Hanford area. Deep drilling at Hanford has shown that two thick basalt layers (one 55 m thick and the other 36 m) occur at about 950 m below the surface that may be suitable for repository construction. Most openings within these layers are filled with alteration products (predominantly clay minerals) and thus provide rock masses of low permeability. These basaltic masses are among the strongest of common rock types. Basalt has moderate thermal conductivity and a high melting temperature; therefore, it can withstand a high thermal load.

Basalts of the Columbia Plateau commonly have zones of columnar joints or rubble that are potential channels for water flow. Water-bearing sedimentary interbeds within the basalt section are also common. The geologic section at Hanford thus comprises a system of alternating aquifers and relatively impermeable zones. The mineralogy and resulting sorptive properties of the partially altered permeable basalt in the sediments must be determined, as they will differ from those of the fresh basalt.

4.3.4 Granite and Related Crystalline Rocks (DOE82, Bu82, K180)

Granite and related crystalline igneous and metamorphic rocks, such as gneiss, have been proposed as potential host rocks for a repository. These are the most abundant rocks in the upper 10 km of the earth's continental crust. Crystalline rocks underlie virtually all of the United States; they occur at the surface in stable areas, in the cores of many mountain ranges, and beneath all of the younger sedimentary cover. Their strength, structural and chemical stability, and low porosity make them attractive for waste repositories. The water content of these rocks is low and is held mainly in fractures and in hydrous silicate minerals.

Because crystalline rocks are ubiquitous, they occur in various tectonic settings. In some areas of the United States, crystalline rocks have been demonstrated to be stable for as long as 2.5 billion years. In other areas, crystalline rocks are involved in younger episodes of mountain building that occurred only tens to hundreds of millions of years ago.

At depths in excess of several hundred meters, where vertical and horizontal stresses increase, the permeability is reduced considerably by closure of the fractures. At some depth, granitic rocks probably become nearly impermeable. A principal goal in evaluating these rocks for nuclear waste disposal will be to use geologic, geophysical, geochemical, and hydrologic investigations to determine the depths at which a repository should be placed so that fracture permeability will not represent an escape pathway for the radionuclides. The safe depth for a repository probably will vary from region to region as a result of the influence of tectonic history on fracture permeability.

4.4 Waste Isolation Pilot Plant (Bu82, DOE80a, DOE83a,b, Le84)

In 1974, DOE began a program to develop a Waste Isolation Pilot Plant in the Los Medanos area of southeastern New Mexico to demonstrate the safe disposal of TRU radioactive wastes from national defense programs. The WIPP, as authorized by Public Law 96-164, is specifically exempted from licensing by the Nuclear Regulatory Commission.

The WIPP is located in a 610-meter-thick bedded salt formation. The formation, which is first encountered at a depth of 260 meters below the surface, is over 200 million years old. The facility has a capacity of 0.18 million cubic meters of contact-handled TRU and 7 thousand cubic meters of remotely handled TRU. The facility, scheduled to begin operation in October 1988, will also contain a research and development area and a retrievable high-level waste experiment area. The limited quantity of high-level waste emplaced for experimental purposes will be removed from the WIPP before the facility is permanently sealed.

4.5 Disposal of DOE Defense High-Level Wastes (NWPA82, DOE84b, DOE85)

The NWPA of 1982 required an evaluation be made to determine the use of disposal capacity at civilian repositories for the disposal of high-level wastes generated by defense activities. The NWPA further states that after factors relating to cost efficiency, health and safety, regulation, transportation, public acceptability, and national security are taken into account, unless the evaluation shows that the development of a separate repository is necessary, the Secretary of Energy shall proceed with arrangements for using the "civilian" repositories for both commercial and defense high-level wastes.

A draft evaluation was prepared by DOE, and because of the cost advantage of disposing of defense wastes in a combined commercial and defense repository, DOE has recommended this option. The NWPA clearly states that costs resulting from permanent disposal of defense high-level waste shall be paid by the Federal government.

4.6 Alternative Disposal Methods (NWPA82, DOE80a,b)

The NWPA also requests the DOE to continue a program of research, development, and investigation of alternative means and technologies for the permanent disposal of high-level radioactive waste from civilian nuclear activities and Federal research and development activities.

Over the years, the DOE and its predecessor agencies have and continue to study several disposal methods. These include:

- Very deep hole concept: placement of containers of waste into holes 3000 to 10,000 meters deep.

- Rock melt concept: placement of fresh liquids or slurries directly into rocks by melting; the heat of the wastes would melt the rock, and thus become incorporated as an integral component of the rock.

- Island-based concept: emplacement of wastes within deep geological formations on a remote isolated island.

- Extraterrestrial concept: disposal of selected fractions of reprocessed waste into an earth escape trajectory or solar orbit.

- Transmutation concept: reduction of selected fractions of reprocessed waste by transmuting it to stable isotopes.

- Well injection concept: disposal of fresh liquid wastes or slurries by deep well injection at depths of 1,000 to 4,800 meters or by shale/grout high-pressure injection at depths of 300 to 480 meters.

- Ice sheet concept: disposal of waste containers in remote continental ice sheets.

- Subseabed concept: emplacement of waste containers on or under the ocean floor. (Present U.S. law and international treaties prohibit disposal of high-level wastes in the ocean.)

4.6.1 In-Place Tank Stabilization (DOE83a,c, Le84, An85)

The DOE is considering the possibility of in-place stabilization of various defense high-level wastes currently stored in single-walled underground tanks if, after the requisite environmental documentation, it is determined that the risks and costs of retrieval and transportation outweigh the environmental benefits of disposal in a mined geologic repository.

The DOE performance assessment for disposal at Hanford of the single-shell tank wastes by in-place stabilization would include stabilizing the waste by drying it to a near solid form or grouting or mixing it with stabilizing chemicals or in situ vitrification, and then covering it with substantial engineered barriers. Current recommended plans call for placing a monument at the surface of the burial sites, and placing permanent records in public libraries, time capsules, computerized information centers, etc., to reduce the probability of all records of the repository being lost.

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Chapter 5: RADIATION DOSIMETRY

5.1 Introduction

Radionuclides transported through the environment may eventually reach people. Contact may occur through either external exposure to radioactively contaminated air, water, and ground surfaces or internal exposure from inhaling or ingesting radioactively contaminated air, water, or food. Individuals in the population may absorb energy emitted by the decaying radionuclides. The quantification of this absorbed energy is termed dosimetry. This chapter describes the dosimetric models for internal and external exposures, the EPA procedure for implementing the dosimetric equations associated with the models, and the uncertainties in dosimetric calculations.

Mathematical models are used to calculate doses to specific human body organs. The models account for the amount of radionuclides entering the body, the movement of radionuclides through the body, and the energy deposited in organs or tissues resulting from irradiation by the radionuclides that reach the tissue. These models provide the basis for the computer codes, RADRISK and DARTAB, which EPA uses to calculate doses and dose rates. (See Appendix A.)

Uncertainties in dosimetric calculations arise from assumptions of uniform distribution of activity in external sources and source organs and assumptions concerning the movement of the radionuclides in the body. The uncertainties associated with dosimetric calculations are difficult to quantify because the data available for determining distribution for the parameters used in the models are usually insufficient. The major source of uncertainty in dosimetry is the real variation in parameter values among individuals in the general population while doses and dose rates are calculated for a "typical" member of the general population. The three sources of dosimetric uncertainty assessed by EPA are: individual variation, age, and measurement errors. The effects of uncertainty are discussed in greater detail in Sections 5.5 and 5.6.

5.2 Definitions

5.2.1 Activity

Radioactive decay is a process whereby the nucleus of an atom emits excess energy. The property possessed by atoms that emit this energy is referred to as radioactivity. The "activity" of a radioactive material is characterized by the number of atoms that emit energy, or disintegrate, in a given period of time. The unit of activity used in this report is the curie (Ci), which equals 3.7×10^{10} disintegrations per second. The excess energy is normally emitted as charged particles moving at high velocities and photons. Although there are many types of emitted radiations, only three are commonly encountered in radioactive material found in the general environment: alpha radiation (nuclei of helium atoms), beta radiation (electrons), and gamma radiation (photons).

The primary mechanism for radiation damage is the transfer of kinetic energy from the moving alpha and beta particles and photons to living tissue. This transfer leads to the rupture of cellular constituents resulting in electrically charged fragments (ionization). Although the amount of energy transferred is small in absolute terms, it is enough to disrupt the molecular structure of living tissue, and, depending on the amount and location of the energy release, leads to the risk of radiation damage.

5.2.2 Exposure and Dose

The term "exposure" herein denotes the subjection of an organ or person to a radiation field. The term "dose" refers to the amount of energy absorbed per gram of absorbing tissue as a result of the exposure. An exposure, for example, may be acute, i.e., occur over a short period of time, while the dose, for some internally deposited materials, may extend over a long period of time.

The dose is a measure of the amount of energy deposited by the alpha and beta particles or photons and their secondary radiations in the organ. The only units of dose used in this chapter are the rad--defined as 100 erg (energy units) per gram (mass unit)--and the millirad (mrad), which is one one-thousandth of a rad. The rad represents the amount, on average, of potentially disruptive energy transferred by ionizing radiation to each gram of tissue. Because it is necessary to know the yearly variation in dose for the calculations described in this report, the quantity used will be the average annual dose (or dose rate) in rad or millirad (per year).

5.2.3 External and Internal Exposures

Radiation doses may be caused by either external or internal exposures. External exposures are those caused by radioactive materials located outside the body, such as irradiation of the body by radioactive material lying on the ground or suspended in the air. Internal exposures are caused by radioactive material that has entered the body through the inhalation or consumption of radioactive material. Having once entered the body, the contaminant may be transmitted to other internal organs and tissues.

The external exposures considered in this report are those resulting from irradiation of the body by gamma rays only. Gamma rays (high energy photons) are the most penetrating of those radiations considered and external gammas may contribute to the radiation dose affecting all organs in the body. Beta particles (electrons), which are far less penetrating, normally deliver their dose to, or slightly below, the unshielded surface of the skin and are not considered because their impact is small, particularly on clothed individuals. Alpha particles (helium nuclei), which are of major importance internally, will not penetrate unbroken skin and so are also excluded from the external dose calculations. The internal exposures considered in this report originate from all three types of radiation.

5.2.4 Dose Equivalent

Different types of charged particles differ in the rate at which their energy is transferred per unit of length traveled in tissue, a parameter called the linear energy transfer (LET) of the particle. Beta particles generally have a much lower LET than alpha particles. Alpha particles are more damaging biologically, per rad, than gamma rays and beta particles. In radiation protection, this difference is accounted for by multiplying the absorbed dose by a factor, Q, the quality factor, to obtain a dose equivalent. The quality factor is intended to correct for the difference in LET of the various particles. At present, the International Commission on Radiological Protection (ICRP) recommends the values $Q=1$ for gamma rays and beta particles and $Q=20$ for alpha particles (ICRP77). The units for the dose equivalent, corresponding to the rad and millirad, are rem and millirem. Thus, dose equivalents for gamma rays and beta particles are numerically equal to the dose since the dose equivalent (mrem) = ($Q=1$) x dose (mrad) while alpha dose equivalents are twenty times as large, dose equivalent (mrem) = ($Q=20$) x dose (mrad).

5.3 Dosimetric Models

The radiation dose has been defined, in Section 5.2.2, as the amount of energy absorbed per unit mass of tissue. Calculation of the dose requires the use of mathematical models such as that shown later in Equation (5-2). In this equation, the amount of activity ingested, I, is multiplied by the fraction, f_1, going to the blood, and the fraction, f_2', going to a specific tissue. E is the amount of energy absorbed by the tissue for each unit of activity so that the product of all these factors divided by the mass of the tissue is, by definition, the radiation dose per unit activity. The remaining term, $[1-e^{-\lambda t}]/\lambda$, indicates how the activity deposited in the tissue changes with time. All these factors together yield the dose rate. A more comprehensive description of the equations used is given in Appendix A.

5.3.1 Internal Doses

Any effort at calculating dose and risk must, of necessity, involve the use of models. In its simplest form, a model is a mathematical representation of a physical or biological system. If, for example, the amount of radioactive material in an organ is measured periodically, a graph of the activity in the organ, such as that in Figure 5.3-1, is obtained. In the simplest case, analysis of these data may indicate that the fraction of the initial activity, R, retained in the organ at any time, t, is given by an equation of the form

$$R = e^{-\lambda t} \tag{5-1}$$

where λ is the elimination rate constant. (More generally, it may require the sum of two or more exponential functions to properly approximate the decrease of radioactivity in the organ. This may be interpreted physically as indicating the existence of two or more "compartments" in the organ from which the radionuclide leaves at different rates.)

The elimination rate constant, λ, is the sum of two terms, which may be measured experimentally, one proportional to the biological clearance half-time and the other proportional to the radioactive half-life. The effective half-life, $t_{1/2}$, for these processes is the time required for one-half of the material originally present to be removed by biological clearance or radioactive decay.

If radionuclides are generally found to follow this behavior, then this equation may be used as a general model for the activity in an organ following deposition of any initial activity. In general, the models used by EPA are those recommended by the ICRP and are documented in detail in the ICRP79. A brief description of each model is given below as an aid to understanding the material presented in the remainder of this chapter.

As mentioned earlier, all radiations--gamma, beta, and alpha--are considered in assessing the doses resulting from internal exposure, that is, exposure resulting from the inhalation or ingestion of contaminated material. Portions of the material inhaled or ingested may not leave the body for a considerable period of time (up to decades); therefore, dose rates are calculated over a corresponding time interval.

The calculation of internal doses requires the use of several models. The most important are the ICRP lung model, depicted in Figure 5.3-2, and the gastrointestinal (GI) tract model shown in Figure 5.3-3. The lung model is comprised of three regions, the nasopharyngial (N-P), the tracheobronchial (T-B), and the pulmonary (P) regions. A certain portion of the radioactive material inhaled is deposited in each of the three lung regions (N-P, T-B, and P) indicated in Figure 5.3-2. The material is then cleared (removed) from the lung to the blood and gastrointestinal tract, as indicated by the arrows, according to the specified clearance parameters for the clearance class of the inhaled material.

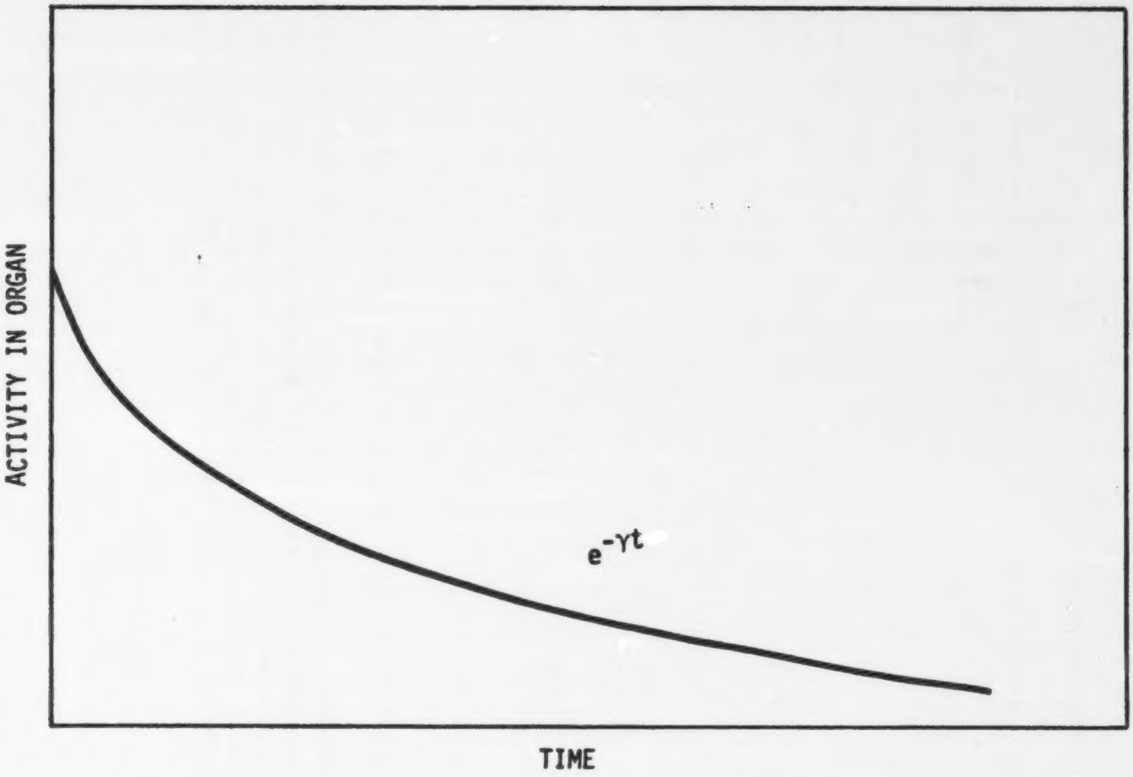

Figure 5.3-1. Typical pattern of decline of activity of a radionuclide in an organ, assuming an initial activity in the organ and no additional uptake of radionuclide by the organ (ORNL81).

Compartment		Class					
		D		W		Y	
		T	F	T	F	T	F
N-P	a	0.01	0.5	0.01	0.1	0.01	0.01
(D_3 = 0.30)	b	0.01	0.5	0.4	0.9	0.4	0.99
T-B	c	0.01	0.95	0.01	0.5	0.01	0.01
(D_4 = 0.08)	d	0.2	0.05	0.2	0.5	0.2	0.99
	e	0.5	0.8	50	0.15	500	0.05
P	f	n.a.	n.a.	1.0	0.4	1.0	0.4
(D_5 = 0.25)	g	n.a.	n.a.	50	0.4	500	0.4
	h	0.5	0.2	50	0.05	500	0.15
L	i	0.5	1.0	50	1.0	1000	0.9

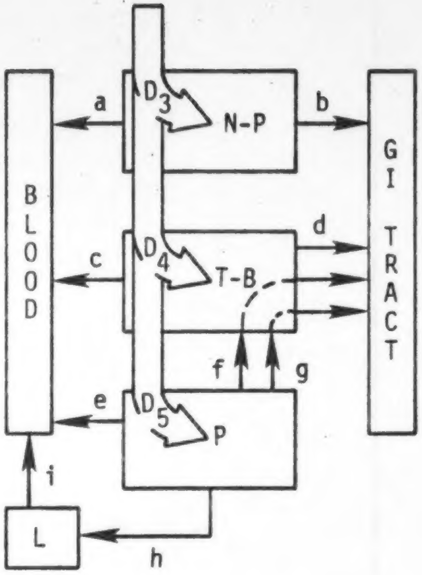

Figure 5.3-2. The ICRP Task Group lung model for particulates.

The columns labeled D, W, and Y correspond, respectively, to rapid, intermediate, and slow clearance of the inspired material (in days, weeks, or years). The symbols T and F denote the biological half-time (days) and coefficient, respectively, of a term in the appropriate retention function. The values shown for D_3, D_4, and D_5 correspond to activity median aerodynamic diameter AMAD = 1 µm and represent the fraction of the inspired material depositing in the lung regions.

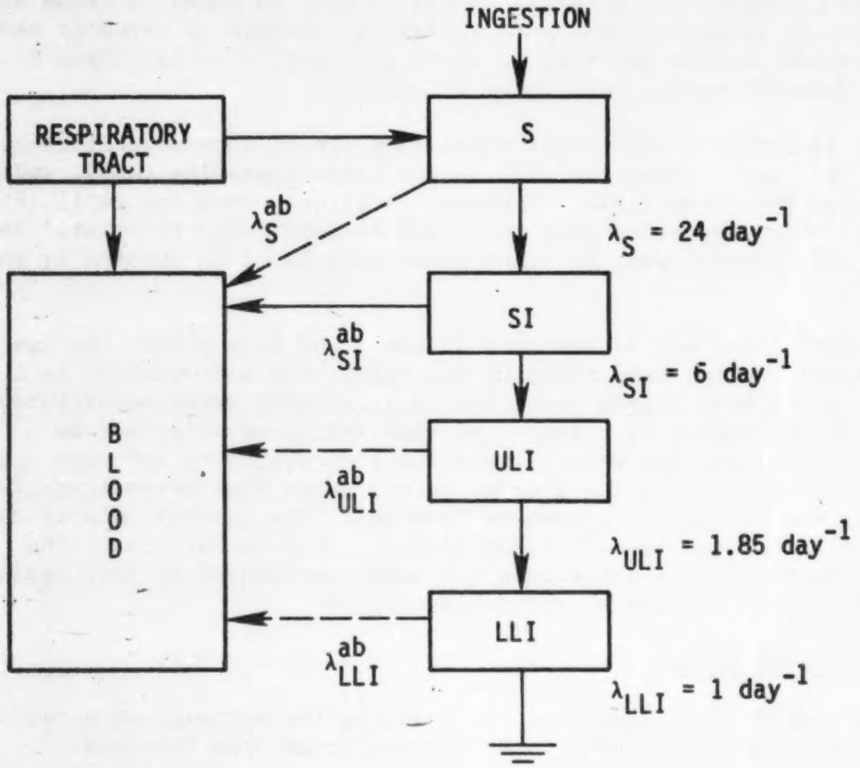
INGESTION
RESPIRATORY TRACT
S
SI
ULI
LLI
B L O O D
λ_S^{ab}
λ_{SI}^{ab}
λ_{ULI}^{ab}
λ_{LLI}^{ab}
$\lambda_S = 24\ \mathrm{day}^{-1}$
$\lambda_{SI} = 6\ \mathrm{day}^{-1}$
$\lambda_{ULI} = 1.85\ \mathrm{day}^{-1}$
$\lambda_{LLI} = 1\ \mathrm{day}^{-1}$

Figure 5.3-3. Schematic representation of radionuclide movement among respiratory tract, gastrointestinal tract, and blood.

S = stomach
SI = small intestine
ULI = upper large intestine
LLI = lower large intestine
λ = elimination rate constant

Deposition and clearance of inhaled materials in the lung are controlled by the particle size and clearance class of the material. The particle size distribution of the airborne material is specified by giving its Activity Median Aerodynamic Diameter (AMAD) in microns, μ, (one micron equals 10^{-6} meters). Where no AMAD is known, a value of 1.0 micron is assumed. Clearance classes are stated in terms of the time required for the material to leave the lung, that is, Class D (days), Class W (weeks), and Class Y (years).

The gastrointestinal tract model consists of four compartments, the stomach (S), small intestine (SI), upper large intestine (ULI), and lower large intestine (LLI). However, it is only from the small intestine (SI) that absorption into the blood is considered to occur. The fraction of material that is transferred into blood is denoted by the symbol f_1.

Radionuclides may be absorbed by the blood from either the lungs or the GI tract. After absorption by the blood, the radionuclide is distributed among body organs according to fractional uptake coefficients, denoted by the symbol f_2'. Since the radioactive material may be transported through the body, dose rates are calculated for each organ or tissue affected by using a model of the organ that mathematically simulates the biological processes involved. The general form of the model for each organ is relatively simple. It postulates that the radioactive material which enters the organ is removed by both radioactive decay and biological removal processes.

5.3.2 External Doses

The example just described for modeling the activity of a radionuclide in an organ pertains to estimating doses from internal exposure. In contrast, the external immersion and surface doses are calculated as follows. First, the number of photons reaching the body is determined. The model used here is a set of equations governing the travel of photons (gamma radiation) in air. The simplifying assumptions used in these calculations are that the medium (air) is an infinite half-space and is the only material present. This makes the calculation relatively straightforward. In the second portion of the calculation, the photons reaching the body are followed through the body using a "Monte Carlo" method. The "phantoms," i.e., the models of the body, are those used by the Medical Internal Radiation Dose Committee (MIRD69). The Monte Carlo method is a procedure in which the known properties of the radiation and tissues are employed to trace (simulate) the paths of a large number of photons in the body. The amount of energy released at each interaction of the radiation with body tissues is recorded and, thus, the dose to each organ or tissue is estimated by evaluating a large number of photon paths.

5.3.3 Effects of Decay Products

In calculating doses from internal and external exposures, the occurrence of radioactive decay products (or daughters) must be considered. When some atoms undergo radioactive decay, the new atom created in the process may also be radioactive and may contribute to the radiation dose. Although these decay products may be treated as independent radionuclides in external exposures, the decay products of each parent must be followed through the body in internal exposures. The decay product contributions to the dose rate are included in the dose calculations, based on the metabolic properties of the element and the organ in which they occur.

5.3.4 Dose Rate Estimates

For each external and internal exposure, dose rates to each of the organs listed in Table 5.3-1 are calculated for each radioisotope. These organ dose rates serve as input to the life table calculations described in Chapter 6.

Table 5.3-1. Organs for which dose rates are calculated

Red bone marrow
Bone
Lung
Breast
Stomach
Pancreas
Intestine
Thyroid
Liver
Urinary tract
Other(a)

(a)Esophagus, lymphatic system, pharynx, larynx, salivary gland, brain.

5.4 EPA Dose Calculation

5.4.1 Dose Rates

The models described in Section 5.2 are used by EPA to calculate radiation dose rates resulting from internal and external exposures to radioactive materials. A more complete description of the methodology, equations, and parameters used is given in Du84, ORNL80, and ORNL81. EPA has adopted two refinements to the ICRP-recommended protocol for these calculations. The first is to track the movement of internally produced radioactive daughters by assuming that their movement is governed by their own metabolic properties rather than those of the parent. Although not enough information is available to allow a rigorously defensible choice, this appears to be more accurate for most organs and radionuclides than the ICRP assumption that daughters behave exactly as the parent. In the second departure from ICRP recommendations, age-dependent values of the parameters governing the uptake of transuranic radionuclides have been taken from two sources deemed appropriate to the general population, the National Radiological Protection Board (NRPB82) and the EPA transuranic guidance document (EPA77).

The internal dose equations given by ICRP may be used to calculate either radiation doses (rad), i.e., the total dose over a given time period, or radiation dose rates (rad/yr), i.e., the way in which the dose changes with time after intake. The integral of the dose rates is, of course, the total dose. EPA calculates dose rates rather than doses, because EPA considers age when assessing the effects of radiation on the population.

External irradiation does not result in any residual internal material. Therefore, external dose rates to a given organ are constant for as long as the external radionuclide is present. That is, the dose rate caused by a given amount of radionuclide present in air or on a ground surface becomes zero when the radionuclide is removed.

The calculation of dose rates, rather than integrated doses, allows the use of age-dependent metabolic parameters more appropriate to the general population to be taken into account. In the vast majority of cases, however, there is not now sufficient information available to make such calculations. The effect of using age-dependent metabolic parameters is discussed in Section 5.2 for some radionuclides for which sufficient information is available.

5.4.2 Exposure and Usage

The ICRP dosimetric equations used by EPA are linear, i.e., an intake of 10 picocuries (pCi) will result in dose rates ten times as large as those from an intake of 1 picocurie. In similar fashion, exposure to ten times as large an air or ground surface concentration will increase the external doses by a factor of ten. EPA uses this linearity to avoid having to calculate radiation dose rates for a range of concentrations. The standard EPA procedure is to use unit intakes of 1 pCi/yr and air and ground surface concentrations of 1 pCi/cm^3 and 1 pCi/cm^2, respectively. The doses for other intakes and concentrations may then be scaled up or down as required.

In most cases, it is necessary to make certain assumptions regarding the exposure conditions in order to perform an assessment. EPA calculates dose rates for lifetime exposure to the unit intakes and concentrations. Chapter 6 describes the different ways in which these rates can be applied. In addition, the exposure assessment will usually depend on other usage conditions assumed for the exposures. For the general population, EPA assumes a breathing rate equal to the ICRP-recommended values (ICRP75), based on 8 hours of heavy activity, 8 hours of light activity, and 8 hours of rest per day. When required, EPA uses a drinking water intake of 2 liters per day. The quantities of food ingested are compiled from a variety of sources. Because there may be insufficient data for some food types, it may be necessary to combine or substitute types in some instances.

5.5 Uncertainty Analysis

Uncertainty, in the dose, refers to the manner in which the calculated dose changes when the parameters used in the calculation (intakes, metabolic factors, organ masses, etc.) are changed. The uncertainty associated with the dosimetric calculations is extremely difficult to quantify because the term "uncertainty analysis" implies a knowledge of parameter distributions that is usually lacking. Internal doses, for example, depend on the parameters used to characterize the physiological and metabolic properties of an individual, while external doses must consider parameters such as organ mass and geometry for a particular individual. The data available for most of these parameters is not sufficient to define the form of the parameter distribution. The major source of uncertainty in calculating the dose to a distinct individual, however, in most instances, does not result from errors in measuring the parameters but from the real variation in parameter values among individuals in the general population. Thus, a calculated dose is thought to be representative of a "typical" member of the general population and is probably reasonably precise for some large segment of that population.

The basic physiological and metabolic data used by EPA in calculating radiation doses are taken from the ICRP Report of the Task Group on Reference Man (ICRP75) and from the ICRP Limits for Intakes of Radionuclides by Workers (ICRP79). The "Reference Man" report is the most comprehensive compilation of data available on the intake, metabolism, internal distribution, and retention of radioisotopes in the human body. Its major purpose, however, is to "define Reference Man, in the first instance, as a typical occupational individual," although differences with respect to age and sex are indicated in some instances.

The limitations inherent in defining Reference Man, and in estimating uncertainties due to variations in individuals in the general population, are recognized by the Task Group (ICRP75):

> "The Task Group agreed that it was not feasible to define Reference Man as an 'average' or a 'median' individual of a specified population group and that it was not necessary that he be defined in any such precise statistical sense. The available data certainly do not represent a random sample of any specified population. Whether the sample is truly representative of a particular population group remains largely a matter of judgement which cannot be supported on the basis of statistical tests of the data since the sampling procedure is suspect. Thus the Task Group has not always selected the 'average', or the 'median', of the available measurements in making its selection, nor has it attempted to limit the sample to some national or regional group and then seek an average or median value. However, the fact that Reference Man is not closely related to an existing population is not believed to be of any great importance. If one did have Reference Man

> defined precisely as having for each attribute the median value of a precisely defined age group in precisely limited locality (e.g., males 18-20 years of age in Paris, France, on June 1, 1964), these median values may be expected to change somewhat with time, and in a few years may no longer be the median values for the specified population. Moreover, the Reference Man so defined would not have this relation to any other population group unless by coincidence. To meet the needs for which Reference Man is defined, this precise statistical relationship to a particular population is not necessary. Only a very few individuals of any population will have characteristics which approximate closely those of Reference Man, however he is defined. The importance of the Reference Man concept is that his characteristics are defined rather precisely, and thus if adjustments for individual differences are to be made, there is a known basis for the dose estimation procedure and for the estimation of the adjustment factor needed for a specified type of individual."

With respect to the dosimetric calculations performed by EPA to assess the impact of radioactive pollutants on a general population, three sources of uncertainty should be considered:

(1) that due to the variation in individual parameters among adults in the general population

(2) that due to the variation in individual parameters with age

(3) that due to experimental error in the determination of specific parameters

Each of these sources of uncertainty is discussed in this section. As noted above, the data required to perform a rigorous uncertainty analysis are lacking, and a form of uncertainty analysis called sensitivity analysis is employed. The sensitivity analysis consists of substituting known ranges in the parameters for the recommended value and observing the resulting change in the calculated dose rate.

5.5.1 Dose Uncertainty Resulting from Individual Variation

This section discusses the uncertainty in calculated radiation doses occasioned by differences in physical size and metabolism among individuals in the general population. In order to investigate the effects of individual differences in intake, size, and metabolism, it is necessary to consider the form of the equation used to calculate radiation dose rates. Equation (5-2) is a simplified form of the one used by EPA to represent the ingestion of radioactive materials.

$$\dot{D}(t) = c\, I\, f_1\, f_2' \, \frac{E}{m} \, \frac{1}{\lambda} \, [1-e^{-\lambda t}] \qquad (5\text{-}2)$$

where $\dot{D}$ is the dose rate (mrem/yr)
I is the intake rate of radioactive material (pCi/yr)
f_1 is the fraction of I transferred to blood after ingestion
f_2' is the fraction transferred to an organ from the blood
m is the mass of the organ (g)
λ is the elimination constant, which denotes how rapidly the activity is removed from the organ (yr^{-1})
E is the energy absorbed by the organ for each radioactive disintegration (ergs)
c is a proportionality constant.

For simplicity, we will assume that dose rates at large times, t, are to be studied so that the term in the bracket is approximately unity.

Although the actual equations used are considerably more complicated because they must describe the lung model and the GI tract, and also treat all radioactive progeny, the essential features of the uncertainty in dose calculations are reflected in the terms of Equation (5-2). The sensitivity of the dose rate to each of the terms in the equation may be studied by substituting observed ranges of the quantities for the single value recommended by Reference Man. For some of these quantities, as noted below, no range is cited because of insufficient data.

Intake, I

As an example, postulate that the ingestion mode to be calculated is for fluid intakes. The average daily fluid intake is about 1900 ml, with an adult range of 1000 to 2400 for "normal" conditions. Under higher environmental temperatures, this range may be increased to 2840 to 3410 ml per day. Thus, a dose calculated as 1.9, for example, could range from 1.0 to 2.4.

Transfer Fraction, f_1

The value of the transfer fraction to blood depends on the chemical form of the element under study. One of the most common naturally occurring radionuclides is uranium, which is used here as an example. ICRP79 cites values of f_1 ranging from 0.005 to 0.05 for industrial workers, but notes that a higher value of 0.2 is indicated by dietary data from persons not occupationally exposed. EPA has used the 0.2 value for the general population but, based on the ICRP range above, a calculated dose determination could vary by a factor of 10.

Organ Mass, m

The range of organ masses depends primarily on the organ under investigation. For example, reported values for the bloodless lungs range from 461 to 676 grams. Liver weights ranged from 1400 to 2300 grams for adult males and 1200 to 1820 grams for females. Thus, because the organ mass appears in the denominator, calculated lung doses might be expected to vary by a factor of 1.5 and liver doses by a factor of about 2.

Remaining Terms, f_2', λ, E

There are few reported data on the ranges in values to be expected for the remaining variables. They are all quantities which are less directly observable than I, f_1, and m and their influence on the dose calculation can only be estimated. The discussion in Section 5.6 is intended to augment the uncertainty analysis by introducing the results of some direct observations on segments of the general population.

5.5.2 Dose Uncertainty Resulting from Age

The dose rates calculated by EPA are normally based on the metabolism and physical characteristics of Reference Man (ICRP75). These properties may obviously be expected to depend on the age of an individual. Most particularly, for infants and children such factors as breathing rates, liquid and solid intakes, organ size and growth rates, and body geometry are known to vary considerably from adult values. The effect of such changes on the radiation dose also depends on the chemistry of the radioactive element under study. For example, rapid bone growth in children is of more importance when a "bone seeker" such as strontium is considered. Although the data available for most age and chemical element combinations are insufficient to allow estimation of the uncertainty in dose rate, some organ/element combinations, for which more information is available, are discussed below.

Iodine and the Thyroid

Iodine is rapidly and virtually completely absorbed into the bloodstream following inhalation or ingestion. From the blood, iodine enters the extracellular fluid and quickly becomes concentrated in the salivary, gastric, and thyroid glands. It is rapidly secreted from the salivary and gastric glands, but it is retained in the thyroid for relatively long periods.

The intake and metabolism of iodine have been reviewed extensively to develop an age-dependent model for iodine (ORNL84a). In the model used here, ingested iodine is assumed to be almost completely absorbed by the blood. The remaining parameters are age dependent and are shown in Table 5.5-1. The fluid intake varies from 0.72 liters per day for a newborn to about 2.0 liters per day for an adult.

These age-dependent parameters may then be used in Equation (5-2) to calculate the dose rate resulting from a constant concentration of iodine in water and air. The resulting curves for the dose rate as a function of age are shown in Figures 5.5-1 and 5.5-2. These may be compared to the dose rates obtained using Reference Man parameters at all ages, indicated by the dotted lines in the same figures. Thus, for this

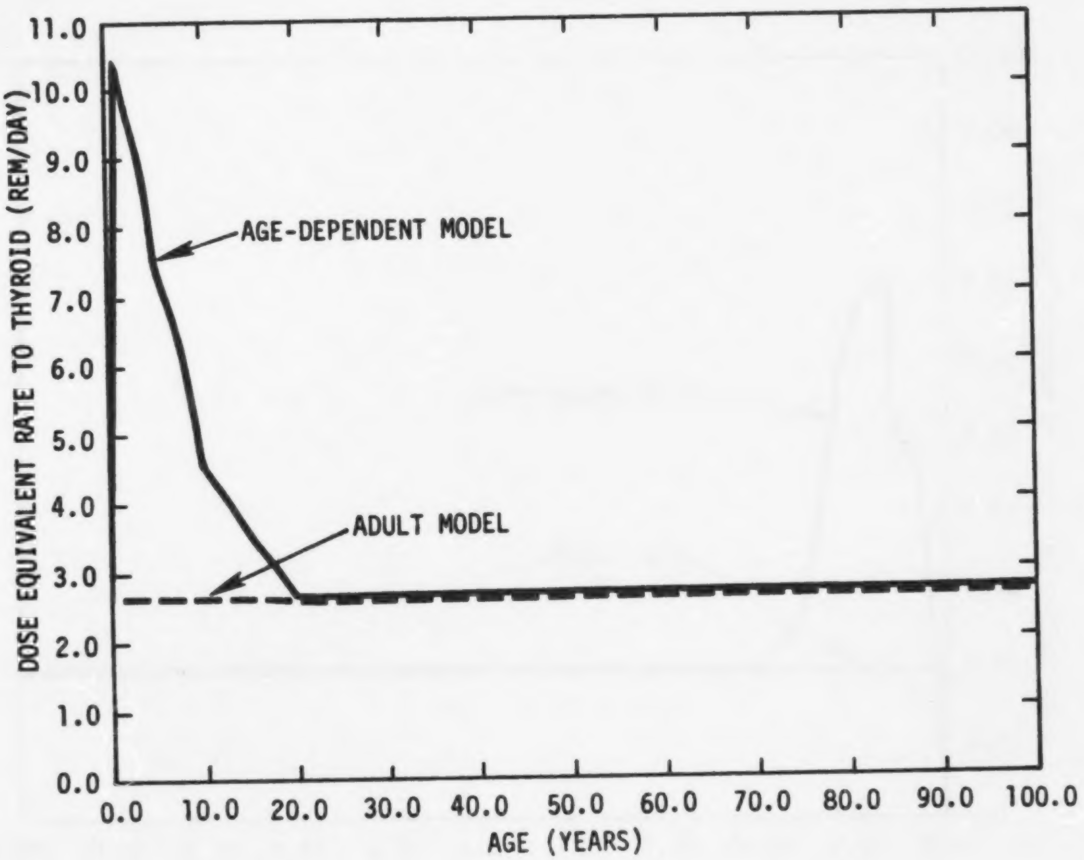

Figure 5.5-1. Dose rate from chronic ingestion of iodine-131 in water at a concentration of 1 μCi/ℓ.

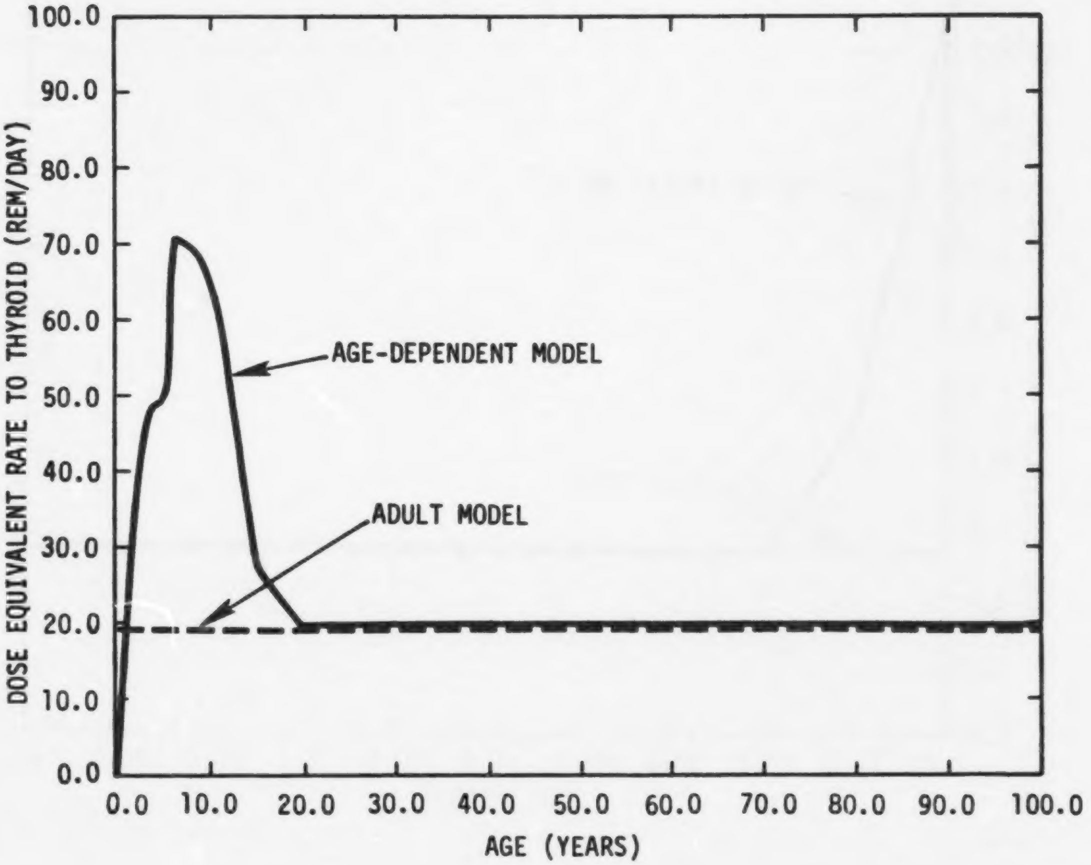

Figure 5.5-2. Dose rate from chronic inhalation of iodine-131 in air at a concentration of 1 μCi/m^3.

particular combination of organ and isotope, the total (70-year) dose is seen to increase by about 30 percent for ingestion and 35 percent for inhalation when dependence on age is considered.

Table 5.5-1. Age-dependent parameters for iodine metabolism in the thyroid

Age (days)	Fractional uptake to thyroid, f_2'	Thyroid mass (g)	Biological half-time in the thyroid (days)
Newborn	0.5	-	15
100	0.4	-	20
365	0.3	1.78	30
1825	0.3	3.45	40
3650	0.3	7.93	50
5475	0.3	12.40	65
7300	0.3	20.00	80

Strontium and Bone

Because of the chemical similarities of strontium and calcium, strontium tends to follow the calcium pathways in the body and deposits to a large extent in the skeleton. In fact, the fraction of ingested strontium eventually reaching the skeleton at a given age depends largely on the skeletal needs for calcium at that age, although the body is able to discriminate somewhat against strontium in favor of calcium after the first few weeks of life.

The ICRP model for bone is more complicated than that for the thyroid because it consists of more than one compartment. For purposes of modeling the transport of strontium by the skeleton, it suffices to view the mineralized skeleton as consisting of two main compartments: trabecular (cancellous, porous, spongy) and cortical (compact) bone. Two subcompartments, surface and volume, are considered within each of these main compartments. The four subcompartments of mineralized skeleton and the movement of strontium among these compartments are shown schematically in Figure 5.5-3. The equations governing the age dependence of the parameters are given in ORNL84a. Dose rate curves for the inhalation and ingestion of constant concentrations of strontium-90 are given in Figures 5.5-4 and 5.5-5. The comparable curves for Reference Man are again indicated by dashed lines. Thus, for this element and organ combination, the dose rate resulting from ingestion is somewhat higher, while the dose rate resulting from inhalation exhibits only minor perturbations, when the age dependence of the parameters is considered. The lifetime (70-year) dose resulting from ingestion is about 7 percent greater and the inhalation dose less than 1 percent different when age dependence is considered.

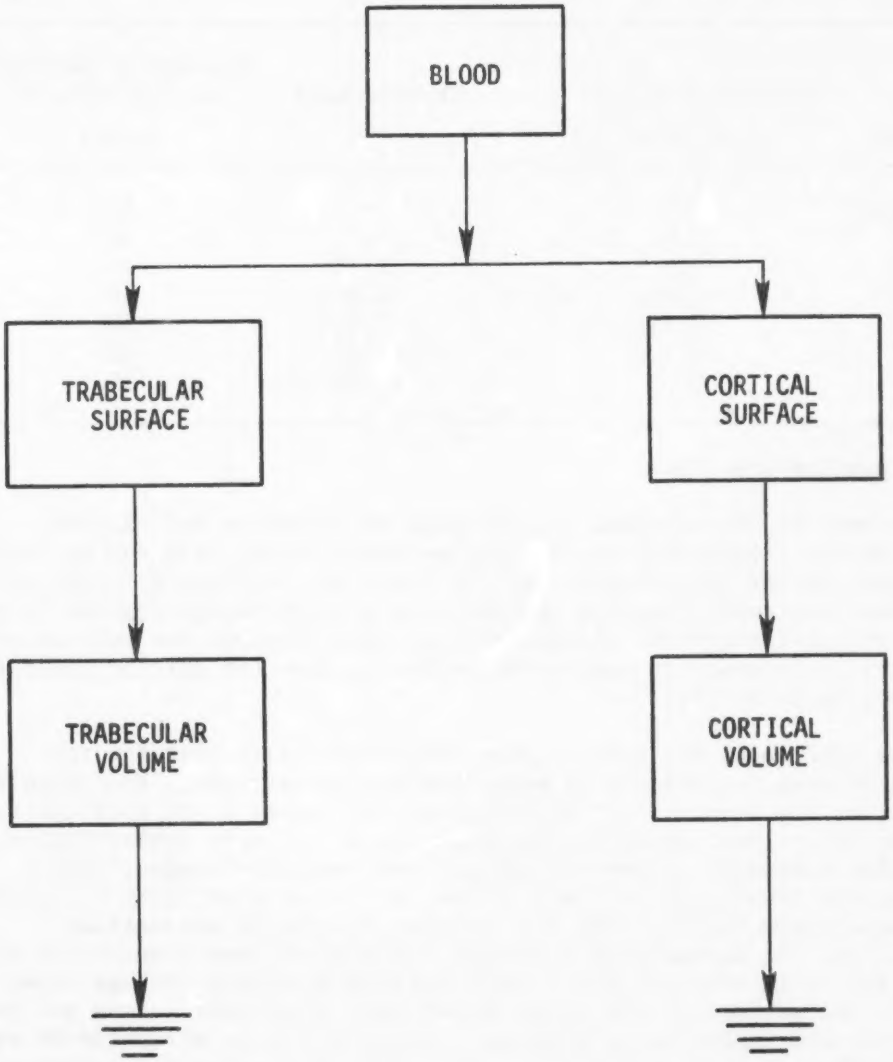

Figure 5.5-3. Compartments and pathways in model for strontium in skeleton.

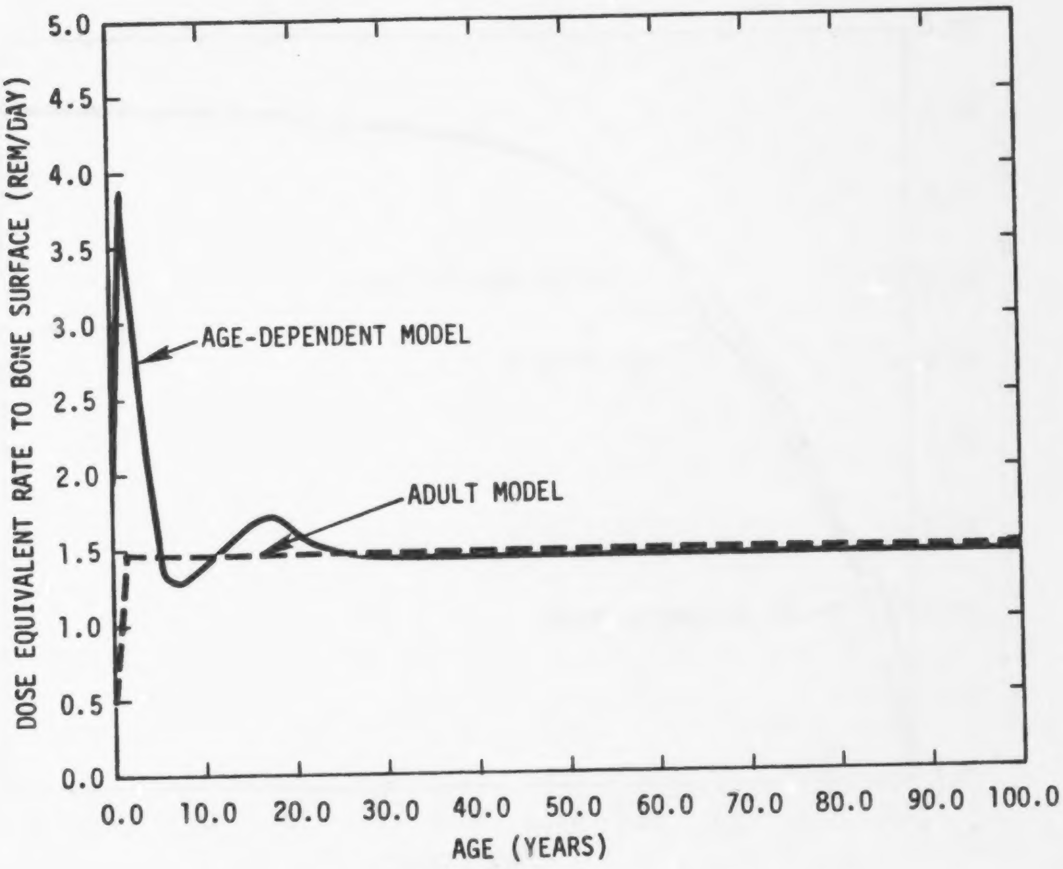

Figure 5.5-4. Dose rate from chronic ingestion of strontium-90 in water at a concentration of 1 μCi/ℓ.

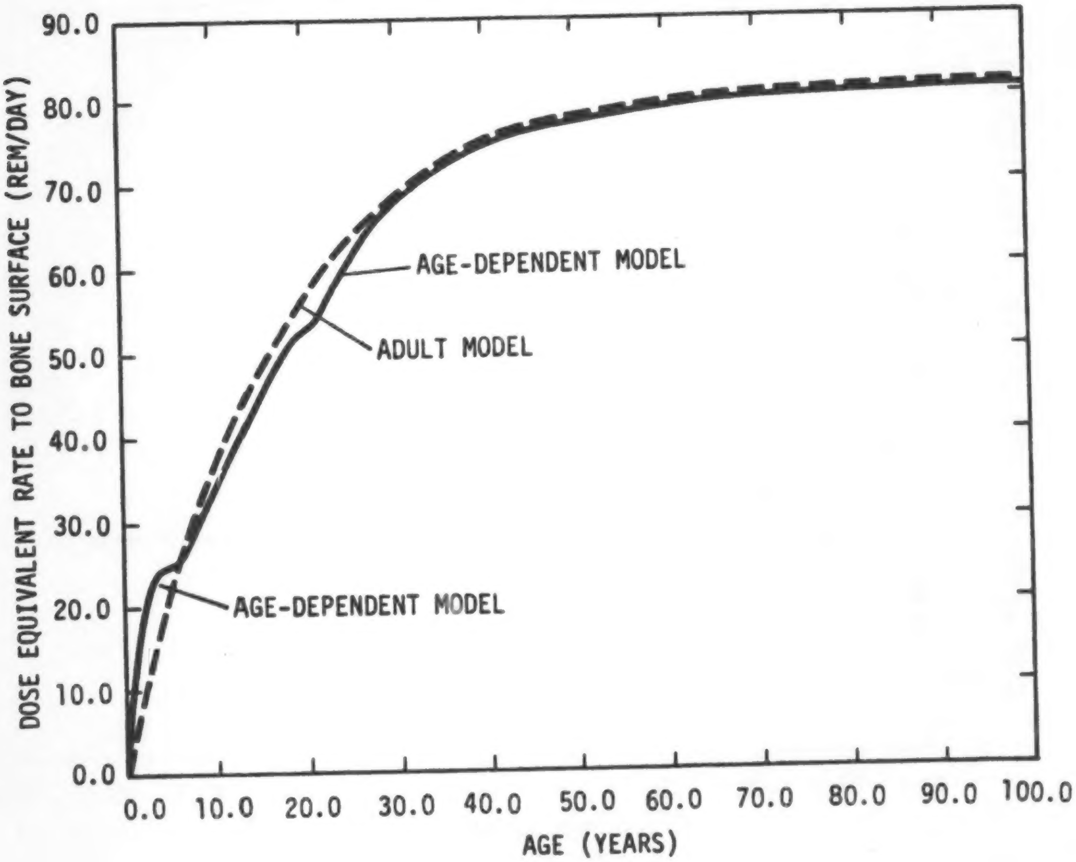

Figure 5.5-5. Dose rate from chronic inhalation of strontium-90 in air at a concentration of 1 $\mu Ci/m^3$.

Plutonium and Lung and Red Bone Marrow

Apparently plutonium and iron bear sufficient chemical resemblance that plutonium is able to penetrate some iron transport and storage systems. It has been shown that plutonium in blood serum complexes with transferrin, the iron-transport protein. Thus, plutonium will partially trace the iron pathway, with the result that a substantial fraction of systemic plutonium is carried to the bone marrow and to the liver. In the skeleton, plutonium may be released mainly at sites of developing red cells. Plutonium that has reached the skeleton behaves very differently from iron; its movement is governed by fairly complicated processes of bone resorption and addition. Because the total metabolic behavior of plutonium is not closely related to that of any essential element, any retention model for plutonium as a function of age will involve much larger uncertainties than the analogous model for strontium. Still, there is enough information concerning the metabolism of plutonium by mammals to justify an examination of potential differences with age in doses to radiosensitive tissues following intake of this radionuclide.

The effect of age-dependent parameters on dose rate calculations is most evident for the lung when the inhalation pathway is considered. Figure 5.5-6 exhibits the variation in dose rate to the total and pulmonary portions of the lung both for the adult and age-dependent cases. The increased dose rate from age 0 to about 20 is typically caused by variations in the breathing rate-lung mass ratio for infants and juveniles. For this model, the age-dependent pulmonary lung 70-year dose is about 9 percent greater than for the adult model.

To describe retention of plutonium in the skeleton, the skeleton is viewed as consisting of a cortical compartment and trabecular compartment. Each of these is further divided into three (rather than two as for strontium) subcompartments: bone surface, bone volume, and a transfer compartment. The transfer compartment, which includes the bone marrow, may receive plutonium that is removed from bone surface or volume; plutonium may reside in this compartment temporarily before being returned either to the bloodstream or to bone surfaces (Figure 5.5-7). Because of the large amount of recycling of plutonium among the skeletal compartments, blood, and other organs, recycling is considered explicitly in the model. The age-dependent features of the model are described in detail in ORNL84a.

Red bone marrow dose rates for the age-dependent model are shown in Figure 5.5-8, for ingestion, and in Figure 5.5-9, for inhalation. The dashed curves are the dose rates using non-age-dependent parameters. As in the corresponding curves for strontium, the difference is more pronounced for the ingestion pathway. Because of the long physical half-life and biological half-time of plutonium in the skeleton, the dose rate, for a chronic intake, does not reach equilibrium within the one hundred year time period of the figures. The total lifetime (70-year) dose to the red marrow is about 25 percent greater for ingestion, and nearly unchanged for inhalation when the age-dependent parameters are used.

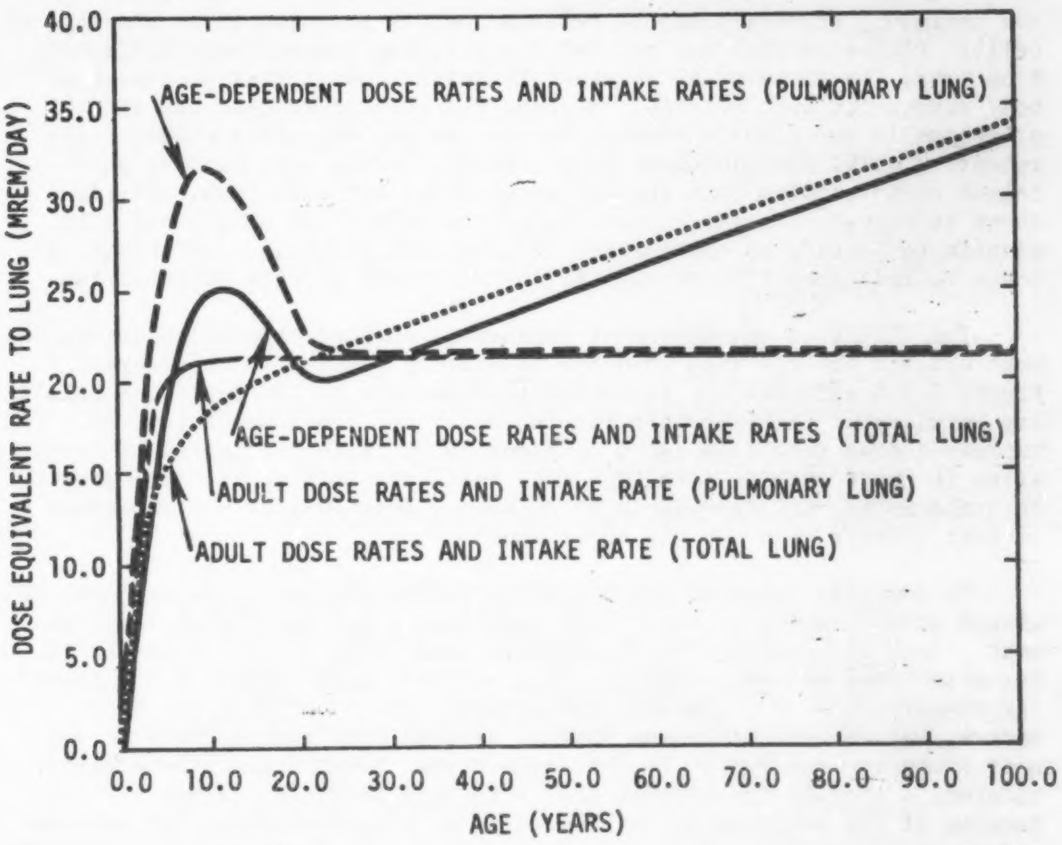

Figure 5.5-6. Dose rate from chronic inhalation of plutonium-239 in air at a concentration of 1 μCi/m^3.

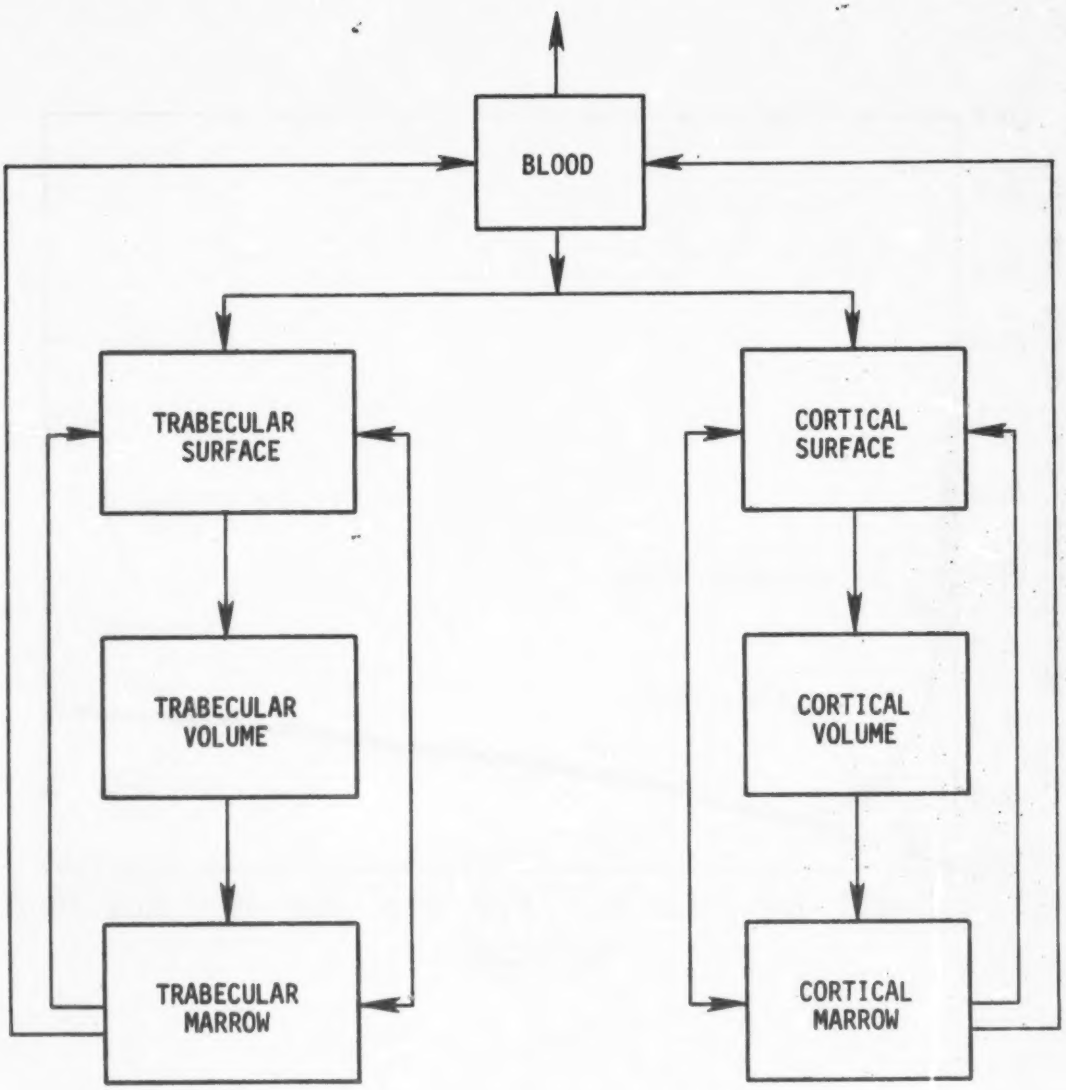

Figure 5.5-7. Compartments and pathways in model for plutonium in skeleton.

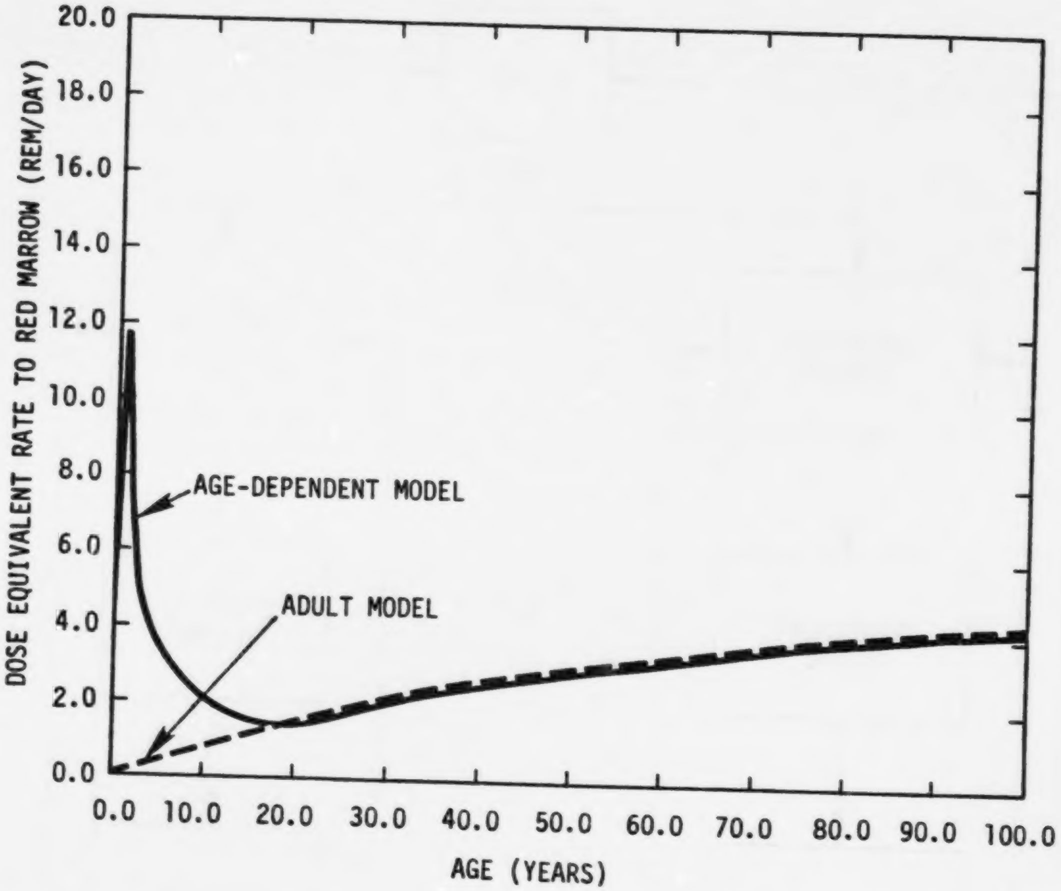

Figure 5.5-8. Dose rate from chronic ingestion of plutonium-239 in water at a concentration of 1 μCi/ℓ.

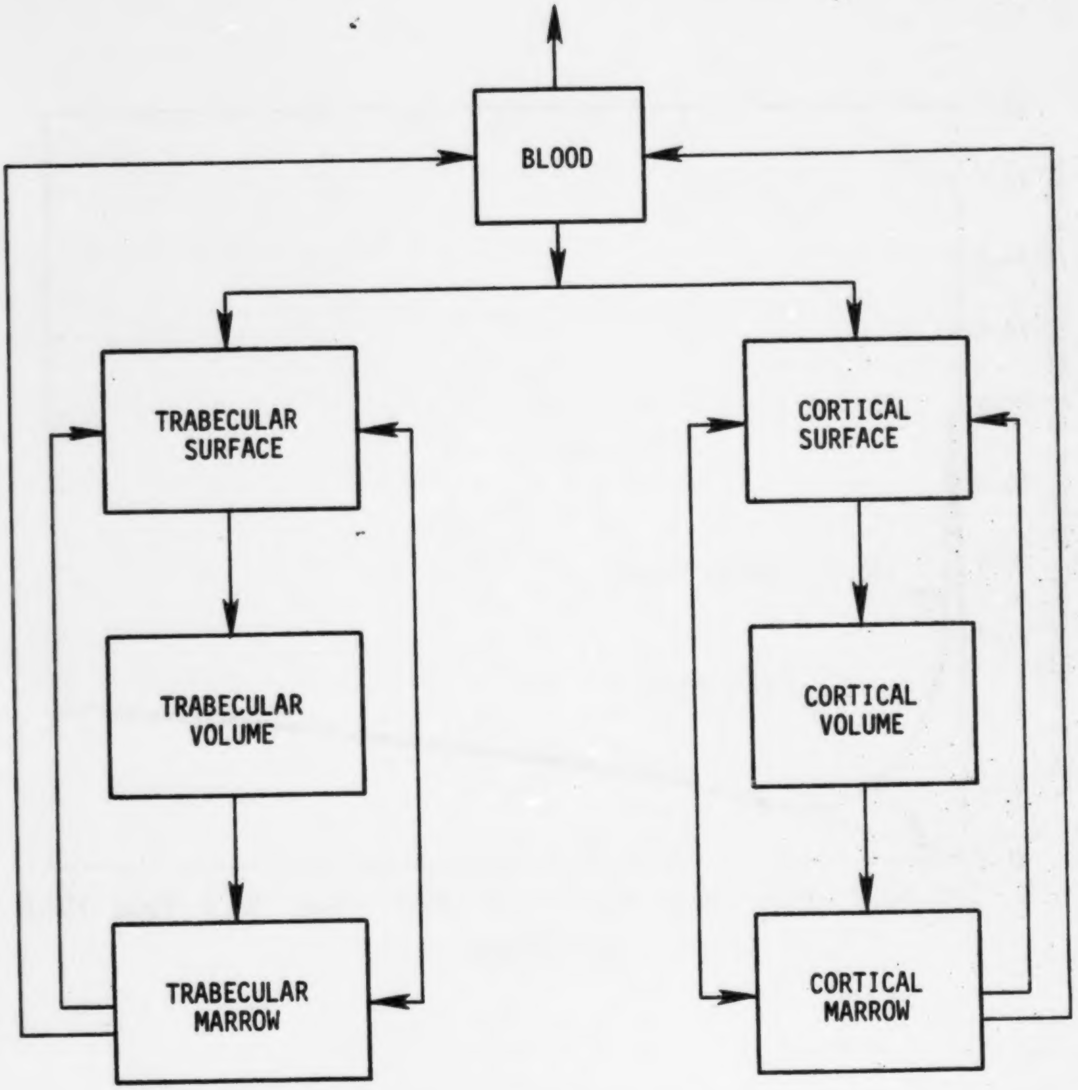

Figure 5.5-7. Compartments and pathways in model for plutonium in skeleton.

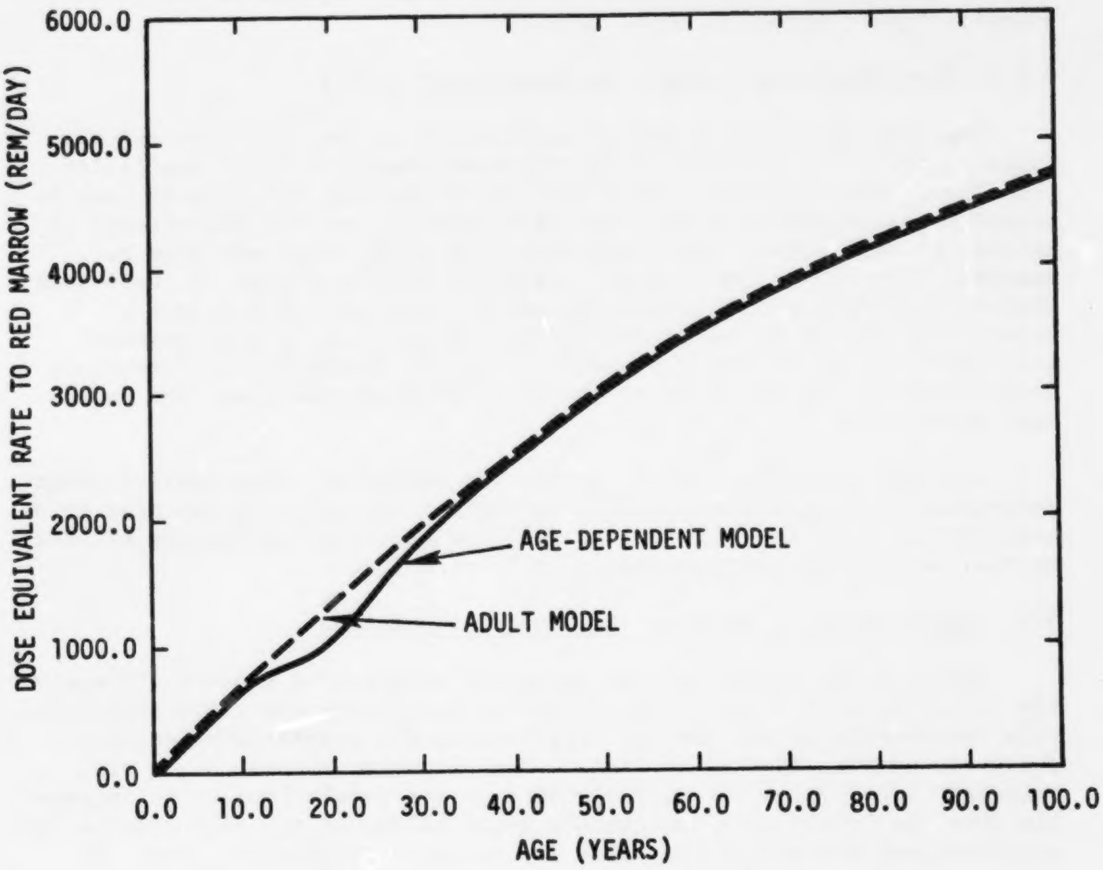

Figure 5.5-9. Dose rate from chronic inhalation of plutonium-239 in air at a concentration of 1 $\mu Ci/m^3$.

In summary, it is difficult to make generalizations concerning the uncertainty involved in neglecting age dependence in the dose rate calculations. Although the examples given indicate higher dose rates for the ingestion pathway, with smaller changes for inhalation, when using age-dependent parameters, this results from the complex interaction between parameters in the dose rate equation and depends on the element/organ combination under consideration.

5.5.3 Dose Uncertainty Caused by Measurement Errors

The last potential source of uncertainty in the dose rate calculations is the error involved in making measurements of fixed quantities (ORNL84b). The radioactive half-life of an isotope, for example, may be measured independently of any biological system, but the measurement is subject to some error. The organ mass of a given organ may also be measured with only a small error. Repeated determinations of these quantities, in addition, can reduce the error. Although this source of uncertainty may be of importance in other aspects of an environmental assessment, it is of little consequence in the dosimetry, because it is overwhelmed by the magnitude of the uncertainties resulting from individual variations.

Although consideration of the factors described above implies large uncertainties in calculated doses, the actual variation is expected to be considerably smaller. The reasons for this, and some supporting studies on real populations, are presented in Section 5.6.

5.6 Distribution of Doses in the General Population

Although the use of extreme parameter values in a sensitivity analysis indicates that large uncertainties in calculated doses are possible, this uncertainty is not usually reflected in the general population. There are several reasons for this: the parameter values chosen are intended to be typical of an individual in the population; it is improbable that the "worst case" parameters would be chosen for all terms in the equation; and not all of the terms are mutually independent, e.g., an increased intake may be offset by more rapid excretion.

This smaller range of uncertainty in real populations is demonstrated by studies performed on various human and animal populations. It should be noted that there is always some variability in observed doses that results primarily from differences in the characteristics of individuals. The usual way of specifying the variability of the dose, or activity, in an organ is in terms of the deviation from the average value. In the following studies, it should also be noted that, in addition to the variability resulting from individual characteristics, the exposure levels of individuals may also have varied appreciably - another factor tending to increase the dose uncertainty. The following studies are representative of those carried out on real populations:

(1) An analysis of the thyroid from 133 jackrabbits in a nuclear fallout area found that in only 2 did the iodine-131 content exceed three times the average (Tu65).

(2) Measurements of the strontium-90 content of adult whole skeletons showed that only about 5 percent of the population would exceed twice the average activity, with only about 0.1 percent exceeding four times the average (Ku62).

(3) In another study, the cesium-137 content of 878 skeletal muscle samples was measured (El64a,b). This radioisotope is also the result of nuclear tests so that the muscle content depends not only on the variation in individual parameters but also on the pathways leading to ingestion or inhalation of the isotope. Nevertheless, analyses of these samples indicated that only 0.2 percent exceeded three times the average activity at a 95 percent confidence level.

(4) A study of the variability in organ deposition among individuals exposed under relatively similar conditions to toxic substances has also been performed (Cu79). In eleven exposure situations (Table 5.6-1), the geometric standard deviation of the apparently lognormal distribution of organ doses ranged from 1.3 to 3.4. From the table, for example, 68 percent of the bone doses resulting from ingestion of strontium-90 would lie between 0.56 and 1.8 times the average.

In all but two of the situations examined, there is the complicating factor that there was probably a great deal of variation in the exposure levels experienced by members of the population. The magnitude of geometric standard deviations of the studies listed in Table 5.6-1 may be the evidence of this variation since, except for the two beagle studies, the exposure was not uniform. Despite these nonuniform exposures, however, the organ dose is not greatly affected probably because of differences in metabolic processes. For example, there is probably some "self-adjustment" in the amount of strontium-90 absorbed from the small intestine to blood of different persons, since strontium-90 tends to vary with calcium in food; if a person has a low calcium intake, then he may absorb a higher fraction of the calcium and strontium-90 than a person with a high calcium intake.

In the beagle studies, the geometric standard deviation is 1.8 for inhaled metals in bone or liver, but is only 1.3 for ingested strontium-90 in bone. An important difference is that all dogs ingesting strontium-90 at a given level were administered the same amount, whereas, in the inhalation studies, the exposure air concentrations were controlled but the dogs inhaled variable amounts depending upon their individual characteristic breathing patterns.

Thus, in real situations, the overall uncertainty in dose is seen to be considerably smaller than would be expected solely on a basis of the "worst case" sensitivity analyses.

Table 5.6-1. Distributions of organ doses[a] from inhalation and ingestion of metals

Population	Exposure	Principal exposure mode	Target organ	Geometric standard deviation of organ doses[a]
Beagle	Metals	Inhalation	Bone or liver	1.8
Humans	Plutonium (fallout)	Inhalation	Lung	3[b]
Humans	Titanium (soil)	Inhalation	Lung	3.4[b]
Humans	Aluminum (soil)	Inhalation	Lung	3.4[b]
Humans	Vanadium (fossil fuel combustion)	Inhalation	Lung	3.4[b]
Beagles	Strontium-90	Ingestion	Bone	1.3
Humans	Strontium-90 (fallout)	Ingestion	Bone	1.8[b]
Humans (smokers)	Cadmium	Inhalation and Ingestion	Kidney	1.8[b]
Humans (nonsmokers)	Cadmium	Inhalation and Ingestion	Kidney	1.8[b]
Humans	Lead	Inhalation and Ingestion	Bone	2.2[b]
Humans	Lead	Inhalation	Lung	1.7[b]

[a] The stable element organ doses used in compiling this table were generally expressed in parts-per-million of organ mass.

[b] Note that exposure levels may vary considerably among individuals; if this factor could be eliminated, geometric standard deviations probably would be smaller.

Source: (Cu79).

5.7 Summary

This chapter presents an overview of the methods used by EPA to estimate radiation doses. The chapter defines the basic quantities reported by EPA and describes briefly the models employed. The chapter also points out departures from the occupational parameters and assumptions employed in the basic ICRP methodology and gives the reasons for the deviations outlined.

Many of the physiological and metabolic parameters recommended in methods for calculating radiation doses are based on a limited number of observations, often on atypical humans or on other species. EPA has attempted to bound the uncertainty associated with the ranges observed for some of the more important parameters used. In fact, some empirical data on population doses mentioned here indicate that actual dose uncertainties are much less than is implied by this "worst case" analysis. For the sources of uncertainty discussed, the large dose ranges possible because of variation in individual characteristics must be modified by consideration of the narrower ranges indicated by studies of real populations; the dose range resulting from age dependence appears to be small for lifetime exposures, and the range resulting from experimental error is negligible by comparison. Based on these observations, it is reasonable to estimate that EPA's calculated doses should be accurate within a factor of 3 or 4. It should be emphasized that much of the "uncertainty" in the dose calculation is not caused by parameter error but reflects real differences in individual characteristics within the general population. Therefore, the uncertainty in the dose estimates cannot be dissociated from specification of the segment of the population to be protected.

More complete derivations and explanations for the EPA methodology are given in the references cited in the text, and a technical description of the dose rate equations and their use in conjunction with the life table risk evaluation is given in Appendix A.

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Chapter 6: ESTIMATING THE RISK OF HEALTH EFFECTS RESULTING FROM RADIONUCLIDES

6.1 Introduction

This chapter describes how EPA estimates the probability of fatal cancer, serious genetic effects, and other detrimental health effects caused by exposure to ionizing radiation. Such risk estimates are complex and uncertain, even though much scientific effort has been expended to increase the understanding of radiation effects.

Because the effects of radiation on human health are known more quantitatively than for most other environmental pollutants, it is possible to make numerical estimates of the risk from a particular source of radioactivity. Such numbers may give an unwarranted aura of certainty to estimated radiation risks. Compared to the baseline incidence of cancer and genetic defects, radiogenic cancer and radiation-induced genetic defects do not occur very frequently. Even among heavily irradiated populations, the number of cancers and genetic defects resulting from radiation is not known with either accuracy or precision simply because of sampling variability. In addition, exposed populations have not been followed for their full lifetime, so that information on ultimate effects is limited. Moreover, when considered in light of information gained from experiments with animals and from various theories of carcinogenesis and mutagenesis, the observational data on the effects of human exposure are subject to a number of interpretations. This in turn leads to differing estimates of radiation risks by both individual radiation scientists and expert groups. Readers should bear in mind that estimating radiation risks is not a mature science and that the evaluation of radiation hazards will change as additional information becomes available. In this chapter, a number of simple mathematical models are presented that may describe the main features of the human response to radiation. However, most scientists would agree that the underlying reality is quite complicated and largely unknown, so that such models should not be taken too literally but rather as useful approximations that will someday be obsolete.

EPA's estimates of cancer and genetic risks in this report are based on the Biological Effects of Ionizing Radiation (BEIR-3) report prepared by the National Academy of Sciences (NAS) Committee in 1980 (NAS80). This report was prepared for the purpose of assessing radiation risks at the low exposure levels of interest in standard setting. As phrased by the President of the Academy, "We believe that the report will be helpful to the EPA and other agencies as they reassess radiation protection standards. It provides the scientific bases upon which standards may be decided after nonscientific social values have been taken into account."

In the sections below, we outline the various assumptions made in calculating radiation risks based on the 1980 NAS report and compare these risk estimates with those prepared by other scientific groups, such as the 1972 NAS BEIR Committee, the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR), and the International Commission on Radiation Protection (ICRP). We recognize that information on radiation risks is incomplete and do not argue that the estimates made by the 1980 NAS BEIR Committee are highly accurate. Rather, we discuss some of the deficiencies in the available data base and point out possible sources of bias in current risk estimates. Nevertheless, we believe the risk estimates made by EPA are "state-of-the-art."

In the sections below, we first consider the cancer risk resulting from whole-body exposure to low-LET* radiation, i.e., lightly ionizing radiation like the energetic electrons produced by X-rays or gamma rays. Environmental contamination by radioactive materials also leads to the ingestion or inhalation of the material and subsequent concentration of the radioactivity in selected body organs. Therefore, the cancer risk resulting from low-LET irradiation of specific organs is examined next. Organ doses can also result from high-LET radiation, such as that associated with alpha particles. The estimation of cancer risks for situations where high-LET radiation is distributed more or less uniformly within a body organ is the third situation considered, Section 6.3. In Section 6.4, we review the causes of uncertainty in the cancer risk estimates and the magnitude of this uncertainty so that the public as well as EPA decision makers have a proper understanding of the degree of confidence to place in them. In Section 6.5, we review and quantify the hazard of deleterious genetic effects from radiation and the effects of exposure in utero on the developing fetus. Finally, in Section 6.6, we calculate cancer and genetic risks from background radiation using the models described in this chapter.

6.2 Cancer Risk Estimates for Low-LET Radiations

Most of the observations of radiation-induced carcinogenesis in humans are on groups exposed to low-LET radiations. These groups include the Japanese A-bomb survivors and medical patients treated with X-rays for ankylosing spondylitis in England from 1935 to 1954 (Sm78). The UNSCEAR and the NAS BEIR-3 Committee have provided knowledgeable reviews of these and other data on the carcinogenic effects of human exposures (UNSCEAR77, NAS80).

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*Linear Energy Transfer (LET) -- the energy deposited per unit of distance along the path of a charged particle.

The most important epidemiological data base on radiogenic cancer is the A-bomb survivors. The Japanese A-bomb survivors have been studied for more than 38 years and most of them, the Life Span Study Sample, have been followed in a carefully planned and monitored epidemiological survey since 1950 (Ka82, Wa83). They were exposed to a wide range of doses and are the largest group that has been studied. Therefore, they are virtually the only group providing information on the response pattern at various levels of exposure to low-LET radiation. Unfortunately, the doses received by various individuals in the Life Span Study Sample are not yet known accurately. The 1980 BEIR Committee's analysis of the A-bomb survivor data was prepared before bias in the dose estimates for the A-bomb survivors (the tentative 1965 dose estimates, T65) became widely recognized (Lo81). It is now clear that the T65 doses tended to be overestimated so that the BEIR Committee's estimates of the risk per unit dose are likely to be too low (Bo82, RERF83,84). A detailed reevaluation of current risk estimates is indicated when the A-bomb survivor data have been reanalyzed on the basis of new and better estimates of the dose to individual survivors.

Uncertainties in radiation risk estimates do not result just from the uncertainties in the Japanese data base and in other epidemiological studies. Analyses of these data bases require a number of assumptions that have a considerable effect on the estimated risk. These assumptions are discussed below.

6.2.1 Assumptions Needed to Make Risk Estimates

A number of assumptions must be made about how observations at high doses should be extrapolated to low doses and low dose rates for radiation of a given type i.e., high- or low-LET (LET). These assumptions include the shape of the dose response function and possible dose rate effects. A dose response function expresses the relationship between dose and the probability that a radiogenic cancer is induced. Observed excess cancers have occurred, for the most part, following relatively high doses of ionizing radiation compared to those likely to occur as a result of the combination of background radiation and environmental contamination from controllable sources of radiation. Therefore, a dose response model provides a method of interpolating between the number of radiogenic cancers observed at high doses and the number of cancers resulting from all causes including background radiation.

The range of interpolation is not the same for all kinds of cancer because it depends upon the radiosensitivity of a given tissue. For example, the most probable radiogenic cancer for women is breast cancer. As described below, breast cancer appears not to be reduced when the dose is delivered over a long period of time. For example, the number of excess cancers per unit dose among Japanese women who received acute doses, is about the same per unit dose as women exposed to small periodic doses of X-rays over many years. If this is actually the case, background radiation is as carcinogenic for breast tissue as the acute exposures from A-bomb gamma radiation. Moreover, the female A-bomb survivors show an excess of breast cancer at doses below 20 rads which is linearly proportional to that observed at several hundred rads (To84). Women in their 40's, the youngest age group in which breast cancer is common, have received about 4 rads of whole-body low-LET background radiation and usually some additional dose incurred for diagnostic medical purposes. Therefore, for this cancer, the difference between observed dose producing radiogenic cancer, less than 20 rads, and the dose resulting from background radiation is less than a factor of five, not several orders of magnitude as is sometimes claimed. However, it should be noted that breast tissue is a comparatively sensitive tissue for cancer induction and that for most cancers, a statistically significant excess has not been observed at doses below 100 rads, low-LET. Therefore, the range of dose interpolation between observed and calculated risk is often large.

6.2.2 Dose Response Functions

The 1980 NAS report examined three dose response functions in detail: (1) linear, in which effects are directly proportional to dose at all doses; (2) linear quadratic, in which effects are very nearly proportional to dose at very low doses and proportional to the square of the dose at high doses; and (3) a quadratic dose response function, where the risk varies as the square of the dose at all doses (NAS80).

We believe the first two of these functions are compatible with most of the data on human cancer. Information which became available only after the BEIR-3 report was published indicates that a quadratic response function is inconsistent with the observed excess risk of solid cancers at Nagasaki, where the estimated gamma-ray doses are not seriously confounded by an assumed neutron dose component. The chance that a quadratic response function underlies the excess cancer observed in the Nagasaki incidence data has been reported as only 1 in 10,000 (Wa83). Although a quadratic response function is not incompatible with the Life Span Study Sample data on leukemia incidence at Nagasaki, Beebe and others have pointed out how unrepresentative these data are of the total observed dose response for leukemia in that city (Be78, El77). There is no evidence that a quadratic response function provides a better fit to the observed leukemia excess among all A-bomb survivors in the Life Span Study Sample than a simple linear model (NAS80). Based on these considerations, we do not believe a quadratic response can be used in a serious effort to estimate cancer risks due to ionizing radiation. EPA notes that neither the National Council on Radiation Protection and Measurements (NCRP), the ICRP, nor other authoritative scientific groups, e.g., National Radiological Protection Board (NRPB) and UNSCEAR, have used a quadratic response function to estimate the risks due to ionizing radiation.

The 1980 NAS BEIR Committee considered only the Japanese mortality data in their analysis of possible dose response functions. Based on the T65 dose estimates, this Committee showed that the excess incidence of solid cancer and leukemia among the A-bomb survivors is compatible with either a linear or linear quadratic dose response to the low-LET radiation component and a linear response to the high-LET neutron component (NAS80). Although the 1980 BEIR report indicated low-LET risk estimates based on a linear quadratic response were "preferred" by most of the scientists who prepared that report, opinion was not unanimous, and we believe the subsequent reassessment of the A-bomb dose seriously weakens the Committee's conclusion. The Committee's analysis of dose response functions was based on the assumption that most of the observed excess leukemia and solid cancers among A-bomb survivors resulted from neutrons. Current evidence, however, is conclusive that neutrons were only a minor component of the dose in both Hiroshima and Nagasaki (Bo82, RERF83,84). Therefore, it is likely that the linear response attributed to neutrons was caused by the gamma dose, not the dose from neutrons. This point is discussed further in Section 6.4.

Reanalysis of the Japanese experience after completion of the dose reassessment may provide more definitive information on the dose response of the A-bomb survivors, but it is unlikely to provide a consensus on the dose response at environmental levels, i.e., about 100 mrads per year. This is because at low enough doses there will always be sampling variations in the observed risks so that observations are compatible, in a statistical sense, with a variety of dose response functions. In the absence of empirical evidence or a strong theoretical basis, a choice between dose response functions must be based on other considerations.

Although there is evidence for a nonlinear response to low-LET radiations in some, but not all, studies of animal radiocarcinogenesis (see below), we are not aware of any data on human cancers that are incompatible with a simple linear model. In such a case, it may be preferable to adopt the simplest hypothesis that adequately models the observed radiation effect. Moreover, EPA believes that risk estimates, for the purpose of assessing radiation impacts on public health, should be based on scientifically creditable risk models that are unlikely to understate the risk. The linear model fulfills this criteria. Given the current bias in the doses assigned to A-bomb survivors (see Section 6.5.1 below), such an approach seems reasonable, as well as prudent. Therefore, in this chapter, EPA has used the BEIR-3 linear dose response model as one of two dose response models for discussing the risk of radiogenic cancer due to low-LET radiations. For low-LET radiations, we have also included discussions of risk that are based on the BEIR-3 linear quadratic dose response model. While in the dose range of interest (environmental levels) the dose squared term in this model is insignificant, the linear term is about 2.5 times smaller than that in the BEIR-3 linear response model. That is, for the same dose, risk estimates based on the BEIR-3 linear quadratic dose response model are only 40 percent of those based on the BEIR-3 linear model.

Many of the risk estimates needed to evaluate the effect of radionuclide releases must be made on an organ specific basis. The BEIR-3 report provides risk coefficients for individual solid cancers only for the linear model in Tables V-14 and V-15. (Tables identified with a V refer to original tables in NAS80 and are not reproduced in this report). We have therefore divided BEIR-3 organ risk estimates for a linear response by a factor of 2.5 to obtain organ specific linear quadratic risk coefficients.

The underlying basis for a linear quadratic response is thought to be that repair of radiation damage mitigates the effect of small doses of radiation or those which occur over a long time period, the reduced linear term being indicative of this repair. Use of a linear quadratic dose response function, as formulated by the BEIR-3 Committee, is equivalent to the use of a dose rate effectiveness factor (DREF) of 2.5 (see below).

The discussions of both the linear and the linear quadratic dose response models for low-LET radiations are included in this chapter to compare the risk estimates obtained for given doses using both models. The more conservative of these two models is the linear model. We have used this model for the calculation of the fatal cancers per curie released to the accessible environment. This policy was thoroughly reviewed and accepted by the High-Level Radioactive Waste Disposal Subcommittee of the EPA Science Advisory Board (EPA84).

6.2.3 The Possible Effects of Dose Rate on Radiocarcinogenesis

The BEIR-3 Committee limited its risk estimates to a minimum dose rate of 1 rem per year and stated that it "does not know if dose rates of gamma rays and X-rays of about 100 mrad/y are detrimental to man." At dose rates comparable to the annual dose that everyone receives for naturally-occurring radioactive materials, a considerable body of scientific opinion holds that the effects of radiation are reduced. The NCRP Committee 40 has suggested that carcinogenic effects of low-LET radiations may be a factor of from 2 to 10 times less for small doses and dose rates than have been observed at high doses (NCRP80).

The low dose and low dose rate effectiveness factors developed by the NCRP Committee 40 are based on their analysis of a large body of plant and animal data that showed reduced effects at low doses for a number of biological endpoints, including radiogenic cancer in animals, chiefly rodents. However, no data for cancer in humans confirm these findings as yet. A few human studies contradict them. Highly fractionated small doses to human breast tissue are apparently as carcinogenic as large acute doses (NAS80, La80). Furthermore, small acute (less than 10 rads) doses to the thyroid are as effective per rad as much larger doses in initiating thyroid cancer (UNSCEAR77, NAS80). Moreover, the increased breast cancer resulting from chronic low dose occupational gamma ray exposures among British radium dial painters is comparable to, or larger than that expected on the basis of acute high dose exposures (Ba81).

While none of these examples is persuasive by itself, collectively they indicate that it may not be prudent to assume that all kinds of cancer are reduced at low dose rates and/or low doses. However, it may be overly conservative to estimate the risk of all cancers on the basis of the linearity observed for breast and thyroid cancer. The ICRP and the UNSCEAR have used a dose rate effectiveness factor of about 2.5 to estimate the risks from occupational and environmental exposures (ICRP77, UNSCEAR77). Their choice of a DREF is fully consistent with and equivalent to the reduction of risk at low doses obtained by substituting the BEIR-3 linear quadratic response model for their linear model. Use of both a DREF and a linear quadratic model for risk estimation is inappropriate (NCRP80).

The difference between risk estimates obtained with the BEIR-3 linear and linear quadratic dose response models is by no means the full measure of the uncertainty in the estimates of the cancer risk resulting from ionizing radiation. (Section 6.4 below summarizes information on uncertainty). Using two models serves as a reminder that there is more than one creditable dose response model for estimating radiation risks and that it is not known if all radiogenic cancers have the same dose response.

6.2.4 Risk Projection Models

None of the exposed groups have been observed long enough to assess the full effects of their exposures, if, as currently thought, most radiogenic cancers occur throughout an exposed person's lifetime (NAS80). Therefore, another major decision that must be made in assessing the lifetime cancer risk due to radiation is to select a risk projection model to estimate the risk for a longer period of time than currently available observation data will allow.

To estimate the risk of radiation exposure that is beyond the years of observation, either a relative risk or an absolute risk projection model (or a suitable variation) may be used. These models are described at length in Chapter 4 of the 1980 NAS report (NAS80). A relative risk projection model projects the currently observed percentage increase in cancer risk per unit dose into future years. An absolute risk model projects the average observed number of excess cancers per unit dose into future years at risk.

Because the underlying risk of cancer increases rapidly with age, the relative risk model predicts a larger probability of excess cancer toward the end of a person's lifetime. In contrast, the absolute risk model predicts a constant incidence of excess cancer across time. Therefore, given the incomplete data we have now, a relative risk model projects somewhat greater risk than that projected using an absolute risk model.

The National Academy of Sciences BEIR Committee and other scientific groups, e.g., UNSCEAR, have not concluded which projection model is the appropriate choice for most radiogenic cancers. However, evidence is accumulating which favors the relative risk projection model for most solid cancers. As pointed out by the 1980 NAS BEIR Committee,

> "If the relative-risk model applies, then the age of the exposed groups, both at the time of exposure and as they move through life, becomes very important. There is now considerable evidence in nearly all the adult human populations studied that persons irradiated at higher ages have, in general, a greater excess risk of cancer than those irradiated at lower ages, or at least they develop cancer sooner. Furthermore, if they are irradiated at a particular age, the excess risk tends to rise *pari passu* (at equal pace) with the risk of the population at large. In other words, the relative-risk model with respect to cancer susceptibility at least as a function of age, evidently applies to some kinds of cancer that have been observed to result from radiation exposure." (NAS80, p.33)

This observation is confirmed by the Ninth A-bomb Survivor Life Span Study, published two years after the 1980 Academy report. This latest report indicates that, for solid cancers, relative risks have continued to remain constant in recent years while absolute risks have increased substantially (Ka82). Smith and Doll have reached similar conclusions on the trend in excess cancer with time among the irradiated spondylitic patients (Sm78).

Although we believe considerable weight should be given to the relative risk model for most solid cancers (see below), the model does not necessarily give an accurate projection of lifetime risk. The mix of tumor types varies with age so that the relative frequency of some common radiogenic tumors, such as thyroid cancer, decreases for older ages. Land has pointed out that this may result in overestimates of the lifetime risk when they are based on a projection model using summed sites relative risks (La83). While this may turn out to be true for estimates of cancer incidence that include cancers less likely to be fatal, e.g., thyroid, it may not be too important in estimating the lifetime risk of fatal cancers since the incidence of most of the common fatal cancers, e.g., breast and lung cancers, increases with age.

Leukemia and bone cancer are exceptions to the general validity of a lifetime expression period for radiogenic cancers. Most, if not all, of the leukemia risk has apparently already been expressed in both the A-bomb survivors and the spondylitics (Ka82, Sm78). Similarly, bone sarcoma from acute exposure appears to have a limited expression period (NAS80, Ma83). For these diseases, the BEIR-3 Committee believed that an absolute risk projection model with a limited expression period is appropriate for estimating lifetime risk (NAS80).

Note that, unlike the NAS72 (BEIR-1) report, the BEIR-3 Committee's relative and absolute risk models are age dependent. That is, the risk coefficient changes, depending on the age of the exposed persons. Observation data on how cancer risk resulting from radiation changes with age is sparse, particularly so in the case of childhood exposures. Nevertheless, the explicit consideration of the variation in radiosensitivity with age at exposure is a significant improvement in methodology. It is important to differentiate between age sensitivity at exposure and the age dependence of cancer expression. In general, people are most sensitive to radiation when they are young. In contrast most radiogenic cancers occur late in life, much like cancers resulting from other causes. In this chapter we present risk estimates for a lifetime exposure of equal annual doses. The cancer risk estimated is lifetime risk from this exposure pattern. However, age-dependent analyses using BEIR-3 risk coefficients indicate that the risk from one year of exposure varies by a factor of at least five, depending on the age of the recipient.

6.2.5 Effect of Various Assumptions on the Numerical Risk Estimates

Differences between risk estimates made by using various combinations of the assumptions described above were examined in the 1980 NAS report. Table 6.2-1, taken from Table V-25, shows the range of cancer fatalities that are induced by a single 10-rad dose as estimated using linear, linear quadratic, and quadratic dose response functions and two risk projection models, relative and absolute (NAS80).

As illustrated in Table 6.2-1, estimating the cancer risk for a given risk projection model on the basis of a quadratic as compared to a linear dose response reduces the estimated risk of fatal cancer by a factor of nearly 20. Between the more credible linear and linear quadratic response functions, the difference is less, a factor of about two and a half. For a given dose response model, results obtained with the two projection models, for solid cancers, differ by about a factor of three.

Even though the 1980 NAS analysis estimated lower risks for a linear quadratic response, it should not be concluded that this response function always provides smaller risk estimates. In contrast to the 1980 NAS analysis where the proportion of risk due to the dose squared term (e.g., C_3 in footnote c of Table 6.2-1) was constrained to positive values, the linear quadratic function (which agrees best with Nagasaki cancer incidence data) has a negative coefficient for the dose squared term (Wa83). Although this negative coefficient is small and indeed may not be significant, the computational result is a larger linear term which leads to higher risk estimates at low doses than would be estimated using a simple linear model (Wa83). Preliminarily, the BEIR-3 analyses of the mortality, which were not restricted to positive coefficients of the dose squared terms, yielded similar results.

Differences in the estimated cancer risk introduced by the choice of the risk projection model are also appreciable. As pointed out above, the 1980 NAS analysis indicates that relative risk estimates exceed absolute risk estimates by about a factor of three. However, relative risk estimates are quite sensitive to how the risk resulting from exposure during childhood persists throughout life. This question is addressed in the next section, where we compare risk estimates made by the 1972 and 1980 NAS BEIR Committees with those of the ICRP and UNSCEAR.

Table 6.2-1. Range of cancer fatalities induced by 10 rads of low-LET radiation (Average value per rad per million persons exposed)

Dose response functions	Lifetime risk projection model	
	Relative[a]	Absolute
Linear[b]	501	167
Linear Quadratic[c]	226	77
Quadratic[d]	28	10

[a] Relative risk projection for all solid cancers except leukemia and bone cancer fatalities, which are projected by means of the absolute risk model.

[b] Response R varies as a constant times the dose, i.e., $R=C_1D$.

[c] $R=C_2D+C_3D^2$.

[d] $R=C_4D^2$.

Source: NAS80, Table V-25.

6.2.6 Comparison of Cancer Risk Estimates for Low-LET Radiation

A number of estimates of the risk of fatal cancer following lifetime exposure are compared in Table 6.2-2. Although all of these risk estimates assume a linear response function, they differ considerably because of other assumptions. In contrast with absolute risk estimates, which have increased since the 1972 NAS report (BEIR-1) was prepared, the 1980 NAS BEIR-3 Committee's estimates of the relative risk, as shown in Table 6.2-2, have decreased relative to those in the BEIR-1 report. This illustrates the sensitivity of risk projections to changes in modeling assumptions. In NAS80, the relative risk observed for ages 10 to 19 was substituted for the considerably higher relative risk observed for those exposed during childhood, ages 0 to 9. In addition, the relative risk coefficients used in the BEIR-3 analysis are based on excess cancer in the Japanese A-bomb survivors compared to U.S. population cancer mortality rates. In NAS72, this excess was compared to cancer mortality in Japan. Moreover, the difference introduced by these two changes, particularly the former, is somewhat greater than indicated in the 1980 NAS report. The relative risk estimate attributed to the BEIR-1 Committee in the NAS 1980 report is incorrect. Therefore, two BEIR-1 relative risk estimates are listed in Table 6.2-2: the risk estimate in NAS80 attributed to the BEIR-1 Committee and an estimate which is based on the risk coefficients in NAS72. The BEIR-3 estimate did not use the relative risk coefficient for childhood exposure given in the BEIR-1 report, which for solid cancers is a factor of 10 larger than adult values (p. 171 in NAS72), but rather used the adult risk for all ages including children. The estimate in Table 6.2-2 labeled NAS72 uses the relative risk coefficients actually given in the BEIR-1 report.

Table 6.2-2. A comparison of estimates of the risk of fatal cancer from a lifetime exposure at 1 rad/year (low-LET radiation)

Source of estimate	Cases per 10^6 person rad	Projection model
BEIR-1 (NAS72)(a)	667	Relative Risk
BEIR-1 (NAS80)(b)	568	Relative Risk
BEIR-3 (NAS80)(b)(c)	403	Relative Risk
BEIR-3 (NAS80)(d)	169	Relative Risk
BEIR-3 (NAS80)(b)	158	Absolute Risk
BEIR-1 (NAS80)(b)	115	Absolute Risk
BEIR-3 (NAS80)(d)	67	Absolute Risk
UNSCEAR (UNSCEAR77)(e)	200-300	None. High dose > 100 rads.
UNSCEAR (UNSCEAR77)(e)	75-175	None. Low dose/dose rate.
ICRP (ICRP77)	125	None. Occupational -- low dose/dose rate.
CLM (Ch83)	100-440	UNSCEAR77 -- without A-bomb data

(a) BEIR-1 relative risk model.
(b) Table V-4 in NAS80, linear dose response.
(c) L-L absolute risk model for bone cancer and leukemia; $\overline{\text{L-L}}$ relative risk model for all other cancer.
(d) Table V-4 in NAS80 linear-quadratic dose response.
(e) Paragraphs 317 and 318 in UNSCEAR77.

By comparing the three relative risk estimates in Table 6.2-2, it is apparent that the relative risk estimates are fairly sensitive to the assumptions made as to what extent the observed high relative risk of cancer from childhood exposure continues throughout adult life. The Life Span Study indicates that the high-risk adult cancer caused by childhood exposures is continuing, although, perhaps, not to the extent predicted by the NAS BEIR-1 Committee (Ka82).

The major reason the two sets of risk estimates in Table 6.2-2 differ is because of the underlying assumption in each set. The NAS BEIR estimates are for lifetime exposure and lifetime expression of induced cancers (NAS72,80). Neither the age distribution of the population at risk nor the projection models (if any) have been specified by either the UNSCEAR or the ICRP. UNSCEAR apparently presumes the same age distributions as occurred in the epidemiological studies they cited, mainly the A-bomb survivors, and a 40-year period of cancer expression. The ICRP risk estimates are for adult workers, presumably exposed between ages 18 and 65, and a similar expression period. These are essentially age-independent absolute risk models with less than lifetime expression of induced cancer mortality. For these reasons alone, risks estimated by ICRP and UNSCEAR are expected to be smaller than those made on the basis of the BEIR-3 report.

The last entry in Table 6.2-2 is of interest because it specifically excludes the A-bomb survivor data based on T65 dose estimates (Ch83). The authors reanalyzed the information on radiogenic cancer in UNSCEAR77 so as to exclude all data based on the Japanese experience. Their estimate of fatalities ranges from 100 to 440 per 10^6 person rad for high doses and dose rates. As indicated in Table 6.2-2, this is somewhat greater but comparable to the UNSCEAR estimate, which includes the A-bomb survivor data. The mean number of fatalities given in Ch83 is 270 per 10^6 person-rem, which is nearly identical to the value EPA has used for a linear dose response model--280 fatalities per 10^6 person rad (see below).

6.2.7 EPA Assumptions About Cancer Risks Resulting from Low-LET Radiation

EPA's discussion of radiation risks in this chapter is based on presumed linear and linear quadratic dose response functions. We believe these are the most credible dose response functions for estimating risks to exposed populations. Using the BEIR-3 linear quadratic model is equivalent, at low dose, to using a dose rate effectiveness factor of 2.5. As stated earlier, we have used a linear dose response function for low-LET radiation in computing the fatal cancers per curie released to the accessible environment.

Except for leukemia and bone cancer, where we use a 25-year expression period for radiogenic cancer, we use a lifetime expression period, as was done in the NAS report (NAS80). Because the most recent Life Span Study Report indicates absolute risks for solid cancers are continuing to increase 33 years after exposure, the 1980 NAS Committee choice of a lifetime expression period appears to be well founded (Ka82). We do not believe limiting cancer expression to 40 years (as has been done by the ICRP and UNSCEAR) is compatible with the continuing increase in solid cancers that has occurred among irradiated populations (Ka82). Analyses of the spondylitic data have led others to similar conclusions (Sm78).

To project the number of fatalities resulting from leukemia and bone cancer, EPA uses an absolute risk model, a minimum induction period of 2 years, and a 25-year expression period. To estimate the number of fatalities resulting from other cancers, EPA uses the arithmetic average of absolute and relative risk projection models. For these cancers, we assume a 10-year minimum induction period and expression of radiation-induced cancer for the balance of an exposed person's lifetime after the minimum induction period.

6.2.8 Methodology for Assessing the Risk of Radiogenic Cancer

EPA uses a life table analysis to estimate the number of fatal radiogenic cancers in an exposed population of 100,000 persons. This analysis considers not only death due to radiogenic cancer, but also the probabilities of other competing causes of death which are, of course, much larger and vary considerably with age (Bu81, Co78). Basically, it calculates for ages 0 to 110 the risk of death due to all causes by applying the 1970 mortality data from the National Center for Health Statistics to a cohort of 100,000 persons (NCHS75). Additional information on the details of the life table analysis is provided in Appendix A. It should be noted that a life table analysis is required to use the age-dependent risk coefficients in the BEIR-3 report. For relative risk estimates, we use age-specific cancer mortality data also provided by NCHS (NCHS73). The EPA computer program we use for the life table analysis was furnished to the NAS BEIR-3 Committee by EPA and used by the Committee to prepare its risk estimates. Therefore, we believe that the population base and calculational approach are similar in both the NAS and EPA analyses.

To project the observed risks of most solid radiogenic cancers beyond the period of current observation, we use both absolute and relative risk models, but usually present an arithmetic average based on these projections. Using a single estimate, instead of a range of values, does not mean that our estimate is precise. As indicated in Table 6.2-2, the range of estimated fatal cancers resulting from the choice of a particular projection model and its internal assumptions is about a factor of three. Although we think it is likely that the relative risk model is the best projection model for most solid cancers, it has been tested rigorously only for lung and breast cancer (La78). Until it has more empirical support, we prefer to use an average risk based on both projection models. A second reason for this choice is to avoid overly conservative risk estimates caused by the compounding of multiplicative conservative assumptions.

To estimate the cancer risk from low-LET, whole-body, lifetime exposure with the linear model, we use the arithmetic average of relative and absolute risk projections (the BEIR-3 L-L model) for solid cancers and an absolute risk projection for leukemia and bone cancer (the BEIR-3 L-L model). For a dose to the whole-body, this yields an estimated 280 fatalities per million person rad. For the BEIR-3 linear quadratic model, which is equivalent to assuming a DREF of 2.5, a low-LET whole body dose yields an estimated life risk of about 110 fatalities per million person rad.

These risk estimates are not unduly conservative. More than 235 of the 280 fatalities estimated with the BEIR-3 linear model result from cancers in soft tissues for which we have used the BEIR-3 L-L model. As explained on page 187 of NAS80, the $\overline{\text{L-L}}$ model is not derived from the observed risk of solid cancers alone but rather includes parameters based on the Committee's analysis of the leukemia mortality data. Therefore, as outlined in Section 6.4, the BEIR-3 Committee's analysis of the Japanese leukemia data depended heavily on the assumption that most of the leukemia observed at Hiroshima was caused by neutrons. In contrast, Table V-30 in the BEIR-3 report estimates the risk of cancer incidence in soft tissues directly, without the additional assumptions contained in the BEIR-3 $\overline{\text{L-L}}$ model. By using the weighted incidence mortality ratios given in the Table V-15, the results given in Table V-30 can be expressed in terms of mortality to yield, for lifetime exposure, an absolute risk estimate of about 200 fatalities per 10^6 person rad and about 770 fatalities per 10^6 person rad when a relative risk projection model is used to estimate lifetime risk. The arithmetic mean of the fatalities projected by these two models is almost 500 per 10^6 person rad, more than twice as many fatal soft tissue cancers as predicted by the BEIR-3 $\overline{\text{L-L}}$ model and about 5 times as many as estimated using the BEIR-3 linear quadratic model.

6.2.9 Organ Risks

By a whole-body dose, we mean a uniform dose to every organ in the body. In practice, such exposure situations seldom occur, particularly for ingested or inhaled radioactivity. This section describes how we apportion this risk estimate for whole-body exposure when considering the risks following the exposure of specific organs.

For most sources of environmental contamination, inhalation and ingestion of radioactivity are more common than direct exposure. In many cases, depending on the chemical and physical characteristics of the radioactive material, inhalation and ingestion result in a nonuniform distribution of radioactive materials within the body so that some organ systems receive much higher doses than others. For example, iodine isotopes concentrate in the thyroid gland, and the dose to this organ can be orders of magnitude larger than the average dose to the body.

Fatal Cancer at Specific Sites

To determine the probability that fatal cancer occurs at a particular site, we have performed life table analyses for each cancer type using the information on cancer incidence and mortality in NAS80. For cancer other than leukemia and bone cancer, we used NAS80 Table V-14 (Age Weighted Cancer Incidence by Site Excluding Leukemia and Bone Cancer) and NAS80 Table V-15, which lists the BEIR Committee's estimates of the ratio of cancer fatality to cancer incidence for these various organs. The proportions of leukemia and fatal bone cancer caused by low-LET radiation were estimated using the results given in Tables V-17 and V-20 of NAS80. Normalized results, which give the proportion of fatal cancer caused by radiogenic cancer at a particular site, are listed in Table 6.2-3. As noted above, these proportions are assumed to be the same for the BEIR-3 linear quadratic dose response model.

Table 6.2-3. Proportion of the total risk of fatal radiogenic cancer resulting from cancer at a particular site

Site	Proportion of total risk[(a)]
Lung	0.21
Breast[(b)]	0.13
Red bone marrow[(c)]	0.16
Thyroid	0.099
Bone	0.009
Liver	0.085
Stomach	0.084
Intestines	0.039
Pancreas	0.058
Kidneys and urinary tract	0.025
Other[(d)]	0.11

(a) NAS80--Lifetime exposure and cancer expression; results are rounded to two figures.

(b) Average for both sexes.

(c) Leukemia.

(d) Total risk for all other organs, including the esophagus, lymphatic system, pharynx, larynx, salivary gland, and brain.

Information on the proportion of fatal cancers resulting from cancer at a particular organ is not precise. One reason is that the data in NAS80 (and Table 6.2-3) are based on whole-body exposures, and it is possible that the incidence of radiogenic cancer varies depending on the number of exposed organs. Except for breast and thyroid cancer, very little information is available on radiogenic cancer resulting from exposure of only one region in the body. Another reason is that most epidemiology studies use mortality data from death certificates, which often provide questionable information on the site of the primary cancer. Moreover, when the existing data are subdivided into specific cancer sites, the number of cases becomes small, and sampling variability is increased. The net result of these factors is that numerical estimates of the total cancer risk are more reliable than those for most single sites.

The 1977 UNSCEAR Committee's estimated risks to different organs are shown in Table 6.2-4. For all of the organs, except the breast, a high and low estimate was made. This range varies by a factor of 2 or more for most organs. Other site-specific estimates show a similar degree of uncertainty, and it is clear that any system for allocating the risk of fatal cancer on an organ-specific basis is inexact (Ka82). Table 6.2-5 compares proportional risks by the NAS BEIR-3 Committee, UNSCEAR, and the ICRP. ICRP Report 26 provides organ-specific weights for assessing combined genetic and cancer risks from occupational exposure (ICRP77). In Table 6.2-5, we have renormalized ICRP risks so that they pertain to cancer alone.

Table 6.2-4. UNSCEAR77 estimates of cancer risks at specified sites

Site	Fatalities per person rad	Average per organ rad	Proportion of total risk
Lung	25-50	37.5	0.24
Breast(a)	25	25.0	0.16
Red bone marrow(b)	15-25	20.0	0.13
Thyroid	5-15	10.0	0.065
Bone	2-5	3.5	0.23
Liver	10-15	12.5	0.081
Stomach	10-15	12.5	0.081
Intestines	14-23	18.5	0.12
Pancreas	2-5	3.5	0.023
Kidneys and urinary tract	2-5	3.5	0.023
Other(c)	4-10	7.0	0.046

(a) Average for both sexes.
(b) Leukemia.
(c) Includes esophagus and lymphatic tissues.

Table 6.2-5. Comparison of proportion of the total risk of radiogenic cancer fatalities by body organ

Site	NAS80(a)	UNSCEAR77	ICRP77(b)
Lung	.21	.24	.16
Breast	.13	.16	.20
Red Marrow	.16	.13	.16
Thyroid	.099	.065	.04
Bone	.009	.023	.04
Liver	.085	.081	(.08)(c)
Stomach	.084	.081	(.08)
Intestine	.039	.12	(.08)
Pancreas	.058	.023	(.08)
Kidneys and urinary tract	.025	.023	(.08)
Other	.11(d)	.046	---

(a) Lifetime exposure and cancer expression.
(b) Normalized for risk of fatal cancer (see text).
(c) Five additional organs which have the highest dose are assigned 0.08 for a total of 0.4.
(d) Others include esophagus, lymphatic system, pharynx, larynx, salivary gland, and brain.

Considering that the cancer risk for a particular site is usually uncertain by a factor of 2 or more, as indicated by the range of UNSCEAR estimates in Table 6.2-4, we would not expect perfect agreement in apportionment of total body risks. Table 6.2-5, however, does indicate reasonable agreement among the three sets of estimates considered here.

The differences between the proportions of the total risk of fatal cancer shown in Table 6.2-5 are, for the most part, small in comparison to their uncertainty. We have used the BEIR-3 organ risks in preference to those made by other groups such as UNSCEAR or the ICRP for several reasons. BEIR estimates of organ risk are based on a projection of lifetime risk using age-specific risk coefficients, rather than just observations to date. Moreover, the 1980 BEIR Committee considered cancer incidence data as well as mortality data. This gives added confidence that the diagnostic basis for their estimates is correct. And, finally, because we apply these proportional organ risk estimates to the NAS80 cancer risk estimates for whole-body exposures, we believe it is consistent to use a single set of related risk estimates. The way we have used NAS80 to estimate mortality resulting from cancer at a particular site is outlined in the next section.

6.2.10 Methodology for Calculating the Proportion of Mortality Resulting from Leukemia

Application of NAS80 to particular problems is straightforward but requires some familiarity with the details of that report. In this section we provide sample calculations based on the BEIR-3 linear dose response model for the case of fatal leukemia resulting from irradiation of the bone marrow throughout an average person's lifetime. We then compared this number to the average number of all fatal radiogenic cancers to obtain the proportion due to leukemia (Table 6.2-3).

The NAS80 estimates in Table 6.2-3 differ from the others in that they include both a consideration of age at exposure and a full expression of radiogenic cancer resulting from lifetime exposure. For example, Table V-17 in NAS80 gives explicit age- and sex-dependent mortality coefficients for leukemia and bone cancer together.

The ratio of leukemia to bone cancer fatalities is given by the coefficient in the dose response relationship listed in Table V-17, i.e., 2.24/0.05. For lifetime exposure at a dose rate of one rad per year, Table V-17 lists 3,568 leukemia (and bone) deaths per 10^6 males and 2,709 deaths per 10^6 females (NAS80). Using a male-female birth ratio of 1.05 to 1.0, this averages to 3,149 fatal cancers per million persons in the general population. The total person rads causing these excess fatalities is the product of one rad per year, 10^6 persons, and 70.7 years (the average age of this population at death). Dividing the total number of fatalities by this product yields 44.5 fatalities per 10^6 person rad of which about 43.5 are due to leukemia. As noted above, for total body exposure, the average of the absolute and relative risk projection models yielded 280 premature cancer deaths per 10^6 person rad. Therefore, P, the proportion of the whole-body risk caused by the lifetime risk of a leukemia death due to lifetime exposure of the red bone marrow, is:

$$P = \frac{43.5}{280} = .16 \quad \text{(cf. with Table 6.2-3)} \qquad (6\text{-}1)$$

To obtain the proportional mortality for other cancers, we have used the site-specific, age-dependent risk coefficients in Table V-14 and the mortality ratios in Table V-15 to calculate the risk of fatal cancer from lifetime exposure at one rad per year (for each sex) and proceeded as in the example for leukemia outlined above.

To apply the data shown in Table 6.2-3 to a particular organ, we multiply the average of the relative and absolute lifetime risk estimates for whole body lifetime exposure for a linear dose response, 280 fatalities per 10^6 person rad and 112 fatalities per 10^6 person rad for a linear quadratic response by the proportional mortality for that cancer. For example, using the linear model, a one rad dose (low-LET) to the kidney (urinary tract) resulting from lifetime exposure is estimated to cause a lifetime probability of death caused by radiogenic cancer that is equal to $(.025) \times (280 \times 10^{-6})$ or 7×10^{-6}, i.e., 7 chances in a million.

Iodine-131 has been reported to be only one tenth as effective as X-rays or gamma rays in inducing thyroid cancer (NAS72, NCRP77). For this cancer a linear dose response and a DREF of 10 is used in calculating lifetime probability of death. For example, the risk from a one rad dose to the thyroid from exposure to iodine-131 or iodine-129 is calculated as follows: $(0.099) \times (0.10) \times (280 \times 10^{-6})$ or 2.8×10^{-6}, about 3 chances in a million.

6.2.11 Cancer Risks Due to Age-Dependent Doses

As noted previously in Chapter 5, almost all of the dose models we have used are based on the ICRP "Reference Man." ICRP dosimetric models are appropriate for adult workers and do not take into account differences resulting from the changes in physiological parameters between children and adults, e.g., intake rates, metabolism, and organ size. Although it is difficult to generalize for all radionuclides, in some cases these differences tend to counterbalance each other. For example, the ratio of minute volume to lung mass is relatively constant with age, i.e., within a factor of two, so that the ICRP adult model for insoluble materials provides a reasonably good estimate of the average annual dose throughout life.

An exception is the thyroid where the very young have a relatively high uptake of radioiodine into a gland which is much smaller than the adult thyroid, as noted in Table 5.5-1. This results in a larger childhood dose and an increased risk which persists throughout life. Since this is a worst case situation, we have examined it with some care, using the age-specific risk coefficients for thyroid cancer in Table V-14 of NAS80 and the age-dependent dose model in ORNL84. For iodine-131 ingestion, the estimated lifetime risk is increased by a factor of 1.56 due to the 30 percent increase in lifetime dose over that obtained with the ORNL adult model, c.f. Chapter 5. Results are about the same for inhalation of iodine-131-- the estimated lifetime risk of fatal thyroid cancer is increased by a factor of 1.63 for ORNL's age-dependent dose estimate.

As noted in Chapter 5, use of an age-dependent dosimetry for other radionuclides has yielded much smaller increased doses relative to adult models and therefore has little effect on estimates of lifetime risk. In particular, the lung dose and risk resulting from the inhalation of insoluble alpha particle emitters is nearly unchanged. The lifetime dose for an age-dependent dose model is only 1.09 times greater than that calculated using an adult model (Chapter 5); the lifetime risk of lung cancer for this age-dependent model is a factor of 1.16 greater than we calculate for life exposure with the adult only model.

6.3 Fatal Cancer Risk Resulting from High-LET Radiation

In this section we explain how EPA estimates the risk of fatal cancer resulting from exposure to high-LET radiation. In some cases, ingestion and inhalation of alpha particle emitting radionuclides can result in a relatively uniform exposure of specific body organs by high-LET radiation. Unlike exposures to X-rays and gamma rays where the resultant charged particle flux results in LET's of the order of 0.2 to 2 keV per micron in tissue, 5 MeV alpha particles result in energy deposition at a track average rate of more than 100 keV per micron. High-LET radiation have a larger biological effect per unit dose (rad) than low-LET radiation. How much greater depends on the particular biological endpoint being considered. For cell killing and other readily observed endpoints, the relative biological effectiveness (RBE) of high-LET alpha radiation is often 10 or more times greater than low-LET radiations.

6.3.1 Quality Factors for Alpha Particles

Charged particles have been assigned quality factors, Q, to account for their efficiency in producing biological damage. Unlike an RBE value, which is for a specific and well-defined endpoint, a quality factor is based on an average overall assessment by radiation protection experts of potential harm of a given radiation relative to X or gamma radiation. In 1977, the ICRP assigned a quality factor of 20 to alpha particle irradiation from radionuclides (ICRP77). The reasonableness of this numerical factor for fatal radiogenic cancers at a particular site is not well known, but it is probably conservative for all sites and highly conservative for some.

The dose equivalent, in rem, is the dose, in rad, times the appropriate quality factor for a specified kind of radiation. For the case of internally deposited alpha particle emitters the dose equivalent from a one-rad dose is equal to 20 rem. It should be noted that prior to ICRP Report 26 (ICRP77), the quality factor for alpha particle irradiation was 10. That is, the biological effect from a given dose of alpha particle radiation was estimated to be 10 times that from an acute dose of low-LET X-rays or gamma rays of the same magnitude in rad. The ICRP decision to increase this quality factor to 20 followed from their decision to estimate the risk of low-LET radiations, in occupational situations, on the assumption that biological effects were reduced at low dose rates for low-LET radiation. There is general agreement that dose rate effects do not occur for high-LET (alpha) radiations. The new ICRP quality factor for alpha particles of 20 largely compensates for the fact that their low-LET risks are now based on an assumed dose rate reduction factor of 2.5. This DREF has been addressed in preparing EPA estimates of the risk per rad for alpha particle doses described below, in Section 6.6.3.

In 1980 the ICRP published a task group report "Biological Effects of Inhaled Radionuclides" which compared the results of animal experiments on radiocarcinogenesis following the inhalation of alpha particle and beta particle emitters (ICRP80). The task group concluded that "the experimental animal data tend to support the decision by the ICRP to change the recommended quality factor from 10 to 20 for alpha radiation."

6.3.2 Dose Response Function

In the case of high-LET radiation, a linear dose response is commonly observed in both human and animal studies and the response is not reduced at low dose rates (NCRP80). Some data on human lung cancer indicate that the carcinogenic response per unit dose of alpha radiation is higher at low doses than higher ones (Ar81, Ho81, Wh83); in addition, some studies with animals show the same response pattern (Ch81, Ul82). We agree with the NAS BEIR-3 Committee that, "For high-LET radiation, such as from internally deposited alpha-emitting radionuclides, the linear hypothesis is less likely to lead to overestimates of the risk and may, in fact, lead to underestimates" (NAS80). However, at low doses, departures from linearity are small compared to the uncertainty in the human epidemiological data, and we believe a linear response provides an adequate model for evaluating risks in the general environment.

A possible exception to a linear response is provided by the data for bone sarcoma (but not sinus carcinoma) among U.S. dial painters who have ingested alpha-emitting radium-226 (NAS80). These data are consistent with a dose squared response (Ro78). Consequently, the NAS BEIR-3 Committee estimated bone cancer risk on the basis of both linear and quadratic dose response functions. However, as pointed out in NAS80, the number of U.S. dial painters at risk who received less than 1000 rad was so small that the absence of excess bone cancer at low doses is not statistically significant. Therefore, the consistency of this data with a quadratic (or threshold) response is not remarkable and, perhaps, not relevant to evaluating risks at low doses. In contrast to the dial painter data, the incidence of bone cancer following radium-224 irradiation, observed in spondylitics by Mays and Spiess in a larger sample at much lower doses, is consistent with a linear response (Ma83, NAS80). Therefore, for high-LET radiations EPA has used a linear response function to evaluate the risk of bone cancer.

Closely related to the choice of a dose response function is what effect the rate at which a dose of high-LET radiation is delivered has on its carcinogenic potential. This is a very active area of current research. There is good empirical evidence, from both human and animal studies, that repeated exposures to radium-224 alpha particles is five times more effective in inducing bone sarcomas than a single exposure which delivers the same dose (Ma83, NAS80). The 1980 NAS BEIR Committee took this into account in their estimates of bone cancer fatalities which EPA is using. We do not know to what extent, if any, a similar enhancement of carcinogenicity may occur for other cancers resulting from internally deposited alpha particle emitters. Nevertheless, we believe the ICRP quality factor of 20 is conservative, even at low dose rates.

6.3.3 Assumptions Made by EPA for Evaluating the Dose from Alpha Particle Emitters

We have evaluated the risk to specific body organs by applying the ICRP quality factor of 20 for alpha radiation to the risk estimates for low dose rate low-LET radiation as described above. For some organs this quality factor may be too conservative. Several authors have noted that estimates of leukemia based on a quality factor of 20 for bone marrow irradiation overpredicts the observed incidence of leukemia in persons receiving thorotrast (thorium dioxide) and in the U.S. radium dial painters (Mo79, Sp83). Nevertheless, in view of the paucity of applicable human data and the uncertainties discussed above, the ICRP quality factor provides a reasonable and prudent way of evaluating the risk due to alpha emitters deposited within body organs.

All of EPA risk estimates for high-LET radiations are based on a linear dose response function. For bone cancer and leukemia we use the absolute risk projection model described in the previous section. For other cancers we use the arithmetic average of relative and absolute risk projections.

Table 6.3-1 indicates the Agency's estimates of the risk of fatal cancer due to a uniform organ dose in various organs from internally deposited alpha particles. These estimates are for lifetime doses at a constant dose rate. It was prepared by multiplying the average risk (based on the linear model for a uniformly distributed whole-body dose of low-LET radiation and a dose rate effectiveness factor of 2.5) by a quality factor of 20 and then apportioning this risk by organ, as indicated in Table 6.3-1.

Table 6.3-1. Estimated number of cancer fatalities from a lifetime exposure to internally deposited alpha particle emitters

Site	Proportional risk[(a)]	Fatalities per 10^6 person rad[(b)]
Lung	.21	460
Breast[(c)]	.13	290
Red marrow[(d)]	.16	350
Thyroid	.099	220
Bone[(e)]	.009	20
Liver	.085	190
Stomach	.084	190
Intestine	.039	90
Pancreas	.058	130
Kidneys and urinary tract	.025	55
Other–Sum (total)	.11	250

(a) Proportion of whole body risk from Table 6.2-3.
(b) Rounded to two figures.
(c) Average for both sexes.
(d) Leukemia.
(e) Bone endosteum as defined in ICRP-30 (ICRP79).

This procedure was not followed for bone cancer. As outlined above, the risk estimate for this cancer in the BEIR-3 report is based on data for high-LET (alpha) radiation and a direct estimation of the effect of the alpha radiation per high-LET rad. Some readers may note that the risk estimate in Table 6.3-1, about 20 bone cancer fatalities per 10^6 person rad, is less than the 27 fatalities listed in Table A-27 of NAS80 for alpha particles. This is because the analysis in Appendix A of NAS80, but not Chapter V of that report, assumes that in addition to a 2-year minimum induction period, 27 years are available for cancer expression. This is usually not the case for doses received beyond middle age. Hence, the estimated lifetime risk is smaller when it is based on a life table analysis that considers lifetime exposure in conjunction with death from all causes.

6.4 Uncertainties in Risk Estimates for Radiogenic Cancer

As pointed out in the introduction of this chapter, numerical estimates of risks due to radiation are neither extremely accurate nor precise. A numerical evaluation of radiogenic cancer risks depends both on epidemiological observations and a number of ad hoc assumptions which are largely external to the observed data set. These assumptions include such factors as the expected duration of risk expression and variations in radiosensitivity as a function of age and demographic characteristics. A major assumption is the shape and slope of the dose effects response curve, particularly at low doses where there is little or no epidemological data. In 1971, the BEIR Committee based its estimates of cancer risk on the assumption that effects at low doses are directly proportional to those observed at high doses, the so called linear-nonthreshold hypothesis. As described above in Section 6.2, the BEIR-3 Committee considered three dose response models and indicated a preference for the linear quadratic model. The risk coefficients the BEIR-3 Committee derived for their linear quadratic model, and to a lesser extent their linear model, are subject to considerable uncertainty primarily because of two factors: 1) systematic errors in the estimated doses of the individual A-bomb survivors, and 2) statistical uncertainty because of the small number of cancers observed at various dose levels.

6.4.1 Uncertainty of the Dose Response Models Due to Bias in the A-bomb Dosimetry

Although the BEIR-3 Committee's choice of a linear quadratic response has gained considerable attention, it may not be generally appreciated that the BEIR-3 Committee's numerical evaluations of dose response functions for cancer due to low-LET radiation were based exclusively on the cancer mortality of the A-bomb survivors. Unfortunately, the dosimetry for A-bomb survivors, on which the BEIR-3 Committee relied, has since been shown to have large systematic errors which serve to undermine the analyses made by the Committee. As outlined below, the mathematical analyses made by the Committee were "constrained" to meet certain a priori assumptions. These assumptions have since been shown to be doubtful.

A careful state-of-the-art evaluation of the dose to A-bomb survivors was carried out by investigators from Oak Ridge National Laboratory in the early 1960's (Au67, Au77). The results of these studies resulted in a "T65" dose being assigned to the dose (kerma) in free air at the location of each survivor for both gamma rays and neutrons. A major conclusion of the ORNL study was that the mix of gamma ray and neutron radiations was quite different in the two cities where A-bombing occurred. These results indicated that at Hiroshima, the neutron dose was more important than the gamma dose when the greater biological efficiency of the high-LET radiations produced by neutrons was taken into account. Conversely, the neutron dose at Nagasaki was shown to be negligible compared to the gamma dose for that range of doses where there were a significant number of survivors. Therefore, the 1980 BEIR Committee evaluated the cancer risks to the survivors at Hiroshima on the assumption that the combined effects of gamma rays and particularly neutrons caused the observed cancer response.

Since the BEIR-3 report was published, it has become evident that the organ doses due to neutrons at Hiroshima were overestimated by about an order of magnitude at distances of 1000 to 1500 meters, where most of the irradiated persons survived bomb blast and yet received significant doses. In fact, the neutron doses at Hiroshima are quite comparable to those previously assigned, at similar distances, to Nagasaki survivors (Ke81a,b, RERF83,84). Moreover, there are now grounds to believe the T65 estimates of gamma ray doses in both cities are also incorrect (RERF83,84). While several factors need further evaluation, reduction of the gamma dose to individual survivors due to the local shielding provided by surrounding structures, is significant. The important point, however, is that the overestimate of the neutron dose to the Hiroshima survivors led to the BEIR-3 Committee attributing most of the risk to neutrons rather than gamma-rays. Hence, they underestimated the risk for low-LET radiations by, as yet, an unknown amount.

For their analysis of the A-bomb survivor data, the BEIR-3 Committee expanded the equations for low-LET radiations listed in Section 6.2, Table 6.2-1, to include a linear dose response function for neutrons:

1) $P(d,D) = c_1 d + k_1 D$ (6-2)

2) $P(d,D) = c_2 d^2 + k_2 D$ (6-3)

3) $P(d,D) = c_3 d + c_4 d^2 + k_3 D$ (6-4)

where d is the gamma dose and D is that part of dose due to high-LET radiations from neutron interactions. Note that in equation (6-4) the linear quadratic (LQ) response, has two linear terms, one for neutrons and one for gamma radiation. In analyzing approximately linear data in terms of equation (6-4), the decision as to how much of the observed linearity should be assigned to the neutron or the gamma component, i.e, k_3 and c_3, respectively, is crucial. As shown below, the BEIR-3 Committee attributed most of the observed radiogenic cancer to a linear response from neutron doses which did not occur.

The BEIR-3 Committee's general plan was to examine the dose response for leukemia and for solid cancer separately to find statistically valid estimates of the coefficients $c_1 \ldots c_4$ and $k_1 \ldots k_3$ by means of regression analyses. The regressions were made after the data were weighted in proportion to their statistical reliability; thus, Hiroshima results dominate the analysis. The T65 neutron and gamma doses to individual survivors are highly correlated since both are strongly decreasing functions of distance. This makes accurate determination of the coefficients in equation (6-4) by means of a regression analysis extremely difficult. In addition, there is considerable sampling variation in the A-bomb survivor data due to small sample size which exacerbates the regression problem. Herbert gives a rigorous discussion of these problems for the case of the A-bomb survivors (He83). Because of these and other problems, agreement between the observed response for solid cancers and that predicted by any of the dose response functions examined by the BEIR-3 Committee is not impressive. For example, goodness of fit, based on Chi square, ranges from 0.20 for equation (6-3) to 0.23 for equation (6-4), to 0.30 for equation (6-2) (Table V-11 in NAS80). For leukemia, the goodness of fit between the observed data and that predicted by the regression analysis is better, e.g., 0.49 for equations (6-2) and (6-3) (Table V8 in NAS80).

The Committee analyzed the A-bomb survivor data in two separate sets, i.e., first leukemia and then all cancer excluding leukemia (solid cancers). Their treatment of these two cases was not equivalent. Unlike the analysis of solid cancers, the Committee's analysis of leukemia considered the Nagasaki and Hiroshima data separately. Their approach (p. 342 in NAS80) appears to be based on an unpublished paper by Charles Land and a published report by Ishimaru et al. on estimating the RBE of neutrons by comparing leukemia mortality in Hiroshima to that in Nagasaki (Is79). Unlike the case for solid cancers (see below), the Committee's regression analysis of the leukemia mortality data did provide stable values for all of the coefficients in equation (6-4), and therefore an RBE for neutrons as a function of dose, as well as the ratio of the linear to the dose-squared terms for leukemia induction due to gamma rays, (c_3/c_4).

Estimating the linear quadratic response coefficients for solid cancers proved to be less straightforward. When the BEIR-3 analysis attempted to fit the A-bomb survivor data on solid cancers to a linear quadratic dose response function, they found that the linear response coefficient, c_3 in equation (6-4), varied from zero to 5.6 depending on the dose range considered. Moreover, their best estimate of the coefficient for the dose squared term in equation (6-4), i.e., c_4, was zero, i.e., the best fit yielded a linear response. Therefore, it was decided that the observations on solid cancers were "not strong enough to provide stable estimates of low dose, low-LET cancer risk when analyzed in this fashion," (NAS80, p. 186).

As outlined in the BEIR-3 Report, the Committee decided to use a constrained regression analysis, that is, substitute some of the parameters for equation (6-4) found in their analysis of leukemia deaths to the regression analysis of the dose response for solid cancers. That is, both the neutron RBE at low dose (the ratio of the coefficient k_3 to c_3) and the ratio of c_3 to c_4, as estimated from the leukemia data, were assumed to apply to the induction of fatal solid cancers. Regression analyses that are constrained in this manner can yield much higher estimates of precision than is warranted by the data, as discussed by Land and Pierce (La83). They can also be very misleading. Herbert has discussed this point in detail as it applies to the BEIR-3 regression analysis (He83). The BEIR-3 Committee's substitution of the results of the leukemia regression for the data on solid cancers allowed them to make stable estimates of c_3, c_4, and k_3. These estimates became the basis for the "preferred" linear quadratic risk estimates for solid cancers presented in NAS80, i.e., the LQ-L model, page 187. (The response models for solid cancers that are based on the Committee's constrained regression analysis are designated with a bar in their 1980 report, e.g., $\overline{\text{LQ-L}}$ and $\overline{\text{L-L}}$.)

Given the information discussed above, it is possible to see, at least qualitatively, how the high bias in the estimated T65 neutron dose to the Japanese survivors affects the 1980 BEIR Committee's "preferred" LQ estimates of the risk coefficients for leukemia. The Committee's age-adjusted risk coefficients for leukemia are listed in Table V-8 (NAS80, page 184). For the linear quadratic response, k_3, the neutron risk coefficient is 27.5. Tables A-11 (NAS80, page 341) and V-6 (NAS80, page 152) provide the estimates of neutron and gamma doses to the bone marrow of Hiroshima survivors that were used by the Committee. Substituting these doses in their risk equations (Table V-8) indicates that about 70 percent of the leukemia deaths were ascribed to the neutron dose component then thought to be present at Hiroshima. As noted above, subsequent research indicates that the high-LET dose due to neutrons was actually much smaller.

It is not possible to accurately quantify what effect the Committee's use of these same coefficients had on their analysis of the dose response for solid cancers. Equation V-10 for solid cancers, p. 187 in NAS80, indicates about 60 percent of the solid tumor response was attributed to the T65 neutron dose; but this is a minimum estimate that ignores the effect of the assumed neutron doses on the value of k_3 and the ratio of c_3 to c_4.

The BEIR-3 Committee's $\overline{\text{LQ-L}}$ model assumes an RBE of 27.8 at low doses. In the Committee's $\overline{\text{L-L}}$ linear response model, the assumed RBE is 11.3. Therefore, this linear model is considerably less sensitive to the neutron dose component, assumed by the Committee, than their $\overline{\text{LQ-L}}$ model. For either model, most of the A-bomb survivors' radiogenic cancer was ascribed to the T65 neutron doses at Hiroshima.

There is no simple way of adjusting the 1980 BEIR risk estimates to account for the risk they attributed to neutrons. Adjustment of neutron doses alone is clearly inappropriate, since there is good reason to believe that T65 estimates of the dose due to gamma rays are also subject to considerable change. Moreover, not all of the individuals in a given T65 dose category will, necessarily, remain grouped together after new estimates of neutron and gamma doses are obtained. Both the numerator and denominator in the ratio of observed to expected cases are subject to change and indeed could change in opposite directions, a fact not considered in some preliminary analyses (St81). Nevertheless, it is reasonable to conclude that bias in the estimated neutron doses at Hiroshima has led to considerable uncertainty in the BEIR-3 risk estimates and also to a systematic underestimation of the risk due to low-LET radiations. For this reason we believe that estimates based on the more conservative linear dose response should be given considerable weight *vis a vis* those made using the BEIR-3 linear quadratic models.

6.4.2 Sampling Variation

In addition to the systematic bias in the BEIR-3 risk estimates for low-LET radiation outlined above, the precision of the estimated linear and quadratic risk coefficients in the BEIR-3 report is poor due to statistical fluctuations due to sample size. Recently, Land and Pierce have reevaluated the precision of the BEIR-3 linear quadratic risk estimates to take into account, at least partially, the Committee's use of a constrained regression analysis (La83). This new analysis indicates that for the BEIR-3 LQ-L model for leukemia, the standard deviation of the linear term is nearly as large as the risk coefficient itself (±0.9 compared to a risk coefficient of 1). For the LQ-L model, solid cancer, the standard deviation is ±1.5 compared to a risk coefficient of 1.6.

It is likely that at least part of the uncertainty attributed to sampling variation in the BEIR-3 risk estimates is not due to sample size and other random factors but rather due to the use of incorrect dose estimates for the A-bomb survivors. The correlation of neutron and gamma-ray doses has been a major underlying cause of the uncertainty in regression analysis using the T-65 doses. Analyses of revised data with much smaller neutron doses may result in better precision. At present, we have concluded that the BEIR-3 risk coefficients are uncertain by at least a factor of two, see below, as well as being biased low by an additional factor of two or more.

6.4.3 Uncertainties Arising from Model Selection

In addition to a dose response model, a "transportation model" is needed to apply the risks from an observed irradiated group to another population having different demographic characteristics. A typical example is the application of the Japanese data for A-bomb survivors to western people. Seymore Jablon, (Director of the Medical Follow-up Agency of the National Research Council, NAS) has called this the transportation problem, a helpful designation because it is often confused with the risk projection problem described below. However, there is more than a geographic aspect to demographic characteristics. The transportation problem includes estimating the risks for one sex based on data for another and a consideration of habits influencing health status such as differences between smokers and nonsmokers.

The BEIR-3 Committee addressed this problem in their 1980 report and concluded, based largely on the breast cancer evidence, that the appropriate way to transport the Japanese risk to the U.S. population was to assume that the absolute risk over a given observation period was transferable but that relative risk was not. Therefore, the Committee calculated what the relative risk would be if the same number of excess cancer deaths were observed in a U.S. population having the same age characteristics as the A-bomb survivors. The base line cancer rates in the U.S. and Japan are quite different for some specific cancers so this is a reasonable approach. However, it contains the assumption that while the cancer initiation process is the same in the two countries, the actual number of radiogenic cancers that actually occur is the result of cancer promotion, the latter being a culturally dependent variable.

An alternative approach to solving the transportation problem is that of the 1972 NAS BEIR-1 Committee. This Committee assumed relative risks would be the same in the United States and Japan and transferred the observed percentage increase directly to the U.S. population. We have compared estimates of the lifetime risk for these two treatments of the transportation problem in order to find out how sensitive the BEIR-3 Committee risk estimates are to their assumptions. To do this, we calculated new relative risk estimates for solid cancers based on the age-specific cancer mortality of the Japanese population rather than the U.S. data used by the BEIR-3 Committee. We found that this alternative approach did not have much effect on the estimated lifetime risk of solid radiogenic cancer, i.e., a change of 3 percent for males, and 17 percent for females. We have concluded that the amount of uncertainty introduced by transporting cancer risks observed in Japan to the U.S. population is small compared to other sources of uncertainty in this risk assessment. Base-line leukemia rates are about the same in the countries, so we believe these risks are also "transportable."

The last of the models needed to estimate risk is a risk projection model. As outlined in Section 6.2, such models are used to project what future risks will be as an exposed population ages. For leukemia and bone cancer, where the expression time is not for a full lifetime but rather 25 years, absolute and relative risk projection models yield the same number of radiogenic cancers, but would distribute them somewhat differently by age. For solid cancers, other than bone, the BEIR-3 Committee assumed that radiogenic cancers would occur throughout the lifetime. This makes the choice of projection model more critical, because the relative risk projection yields estimated risks about three times larger than that obtained with an absolute risk projection, as shown in Table 6.2-2. Because we have used the average of these two projections for solid cancers, we believe this reduces the uncertainty from the choice of model to about a factor of two or perhaps less, depending on the age distribution of fatal radiogenic cancer, as outlined in Section 6.2 above.

Similarly, there is as yet insufficient information on radiosensitivity as a function of the age at exposure. The age-dependent risk coefficients we have used are those presented in the BEIR-3 report. As yet, there is little information on the ultimate effects of exposure during childhood. As the A-bomb survivors' population ages, more information will become available on the cancer mortality of persons irradiated when they were young. Table 6.2-2 indicates that the more conservative BEIR-1 estimates for the effect of childhood exposures would increase BEIR-3 risk estimates by about 40 percent. As this is probably an upper limit, the lack of more precise information is not a major source of uncertainty in estimates of the risk caused by lifetime exposure. Similarly, the BEIR-3 Committee did not calculate population risks for radiogenic cancer that included *in utero* radiation because they felt the available data were unreliable. We have deferred to their judgment in this regard. The BEIR-1 report did include *in utero* cancer risk. These had little effect, 1 to 10 percent, on the lifetime risk of cancer from lifetime exposure. An effect this small is not significant relative to other sources of uncertainty in the risk assessment.

6.4.4 Summary

We can only semi-quantitatively estimate the overall uncertainty in the risk per rad for low-LET radiations. We expect that more quantitative estimates of the uncertainty will be possible only after the A-bomb dose reassessment is completed and the A-bomb survivor data reanalyzed on the basis of the new dose estimates. It should be noted, however, that even if all systematic bias is removed from the new dose estimates, there will still be considerable random error in the dose estimate for each survivor. This random error biases the estimated slope of the dose response curve so that it is smaller than the true dose response (Da72, Ma59). The amount of bias introduced depends on the size of the random error in the dose estimates and their distribution which are unknown quantities at this stage of the dose reassessment.

The source of uncertainty in risk estimates for low-LET radiations can be ranked as shown in Table 6.4-1.

Table 6.4-1. A ranking of causes of uncertainty in estimates of the risk of cancer

Source of uncertainty	Degree of uncertainty
Choice of dose response model	±250 percent(a)
Slope of dose response resulting from sampling variation	±200 percent(b)
Choice of an average risk projection model	±100 percent(c)
Choice of transportation model	±20 percent(d)
A-bomb T-65 dosimetry	Plus only, amount unknown

(a)For choices limited to BEIR-3 linear and linear quadratic models, see 6.2.
(b)Estimate of 2 standard deviations for the BEIR-3 $\overline{\text{LQ}}$ model (La83).
(c)Average of relative and absolute projection as described above.
(d)For the total of all cancers, not specific cancers.

The estimates of uncertainty in Table 6.4-1 are not wholly comparable and must be interpreted carefully. However, they do have some illustrative value, particularly when ordered in this way. The uncertainty listed for the slope of dose response is a nominal value for the BEIR-3 linear quadratic LQ formulation (La83) in that it is only valid insofar as the Committee's assumptions are true. It is based on a two standard deviation error so that the expectation that the error is less than indicated is 95 percent. We do not believe the uncertainty in the BEIR-3 linear estimate, L-L, is significantly smaller, c.f. Tables V-9 and V-11 in NAS80.

The other uncertainties listed in Table 6.4-1 are quite different, being more in the nature of informed judgments than the result of a statistical analysis. It is doubtful that all radiogenic cancers have the same type of response functions. However, if they were all linear, as breast cancer and thyroid appear to be, the BEIR-3 linear quadratic response model would underestimate the response by 250 percent. If most cancers have a linear quadratic response, or equivalently, a dose rate reduction factor equal to the difference in slope at low doses between the BEIR-3 linear and linear quadratic models, the use of a linear model would overestimate the response by a factor of 2.5. We believe that a factor of 250 percent is a conservative estimate of the uncertainty introduced by the lack of data at low dose rates.

As discussed above, the uncertainty due to the choice of an absolute or a relative risk model is about a factor of three. The use of the average risk for these two models reduces the uncertainty in risk projection by more than a factor of two, since it is known that a relative risk projection is high for some kinds of cancer and that an absolute risk projection is low for others.

The uncertainties listed in Table 6.4-1 are largely independent of each other and therefore unlikely to be correlated in sign. Their root mean square sum is about 300 percent, indicating the expectation that calculated risks would be within a factor of three or so of the true value. This result is overly optimistic because it does not include consideration of the uncertainty introduced by the bias in the A-bomb dosimetry or by the constrained regression analysis used by the BEIR-3 Committee.

6.5 Other Radiation-Induced Health Effects

The earliest report of radiation-induced health effects was in 1896, and it dealt with acute effects in skin caused by x-ray exposures (Mo67). Within the six-year period following, 170 radiation-related skin damage cases had been reported. Such injury, like many other acute effects, is the result of exposure to hundreds or thousands of rads. Under normal environmental exposure situations, however, such exposure conditions are not possible and therefore will not be considered in assessing the risk to the general population from radionuclide releases.

Although radiation-induced carcinogenesis was the first delayed health effect reported, radiation-induced genetic changes were reported early too. In 1927, H.J. Muller reported on x-ray induced mutations in animals and in 1928, L.J. Stadler reported a similar finding in plants (Ki62). At about the same time, radiation effects on the developing embryo were reported. Case reports in 1929 showed a high rate of microcephaly (small head size) and central nervous system disturbance and one case of skeletal defects in children irradiated in utero (UNSCEAR69). These effects, at unrecorded but high exposures, appeared to produce central nervous system and eye defects similar to those reported in rats as early as 1922 (Ru50).

For purposes of assessing the risks of environmental exposure from radionuclide releases, the genetic effects and in utero developmental effects are the only health hazards other than cancer that are addressed in this BID.

6.5.1 Types of Genetic Harm and Duration of Expression

Genetic harm or the genetic effects of radiation exposure are those effects induced in the germ cells (eggs or sperm) of exposed individuals, which are transmitted to and expressed only in their progeny and future generations.

Of the possible consequences of radiation exposure, the genetic risk is more subtle than the somatic risk. Genetic risk is incurred by fertile people when radiation damages the nucleus of the cells which become their eggs or sperm. The damage, in the form of a mutation or a chromosome aberration, is transmitted to, and may be expressed in, a child conceived after the radiation exposure and in subsequent generations. However, the damage may be expressed only after many generations or, alternately, it may never be expressed because of failure to reproduce.

EPA treats genetic risk as independent of somatic risk because, although somatic risk is expressed in the person exposed, genetic risk is expressed only in progeny and, in general, over many subsequent generations. Moreover, the types of damage incurred often differ in kind from cancer and cancer death. Historically, research on genetic effects and development of risk estimates has proceeded independently of the research on carcinogenesis. Neither the dose response models nor the risk estimates of genetic harm are derived from data on studies of carcinogenesis.

Although genetic effects may vary greatly in severity, the genetic risks considered by the EPA when evaluating the hazard of radiation exposure include only those "disorders and traits that cause a serious handicap at sometime during lifetime" (NAS80). Genetic risk may result from one of several types of damage that ionizing radiation can cause in the DNA within eggs and sperm. The types of damage usually considered are: dominant and recessive mutations in autosomal chromosomes, mutations in sex-linked (x-linked) chromosomes, chromosome aberrations (physical rearrangement or removal of part of the genetic message on the chromosome or abnormal numbers of chromosomes), and irregularly inherited disorders (genetic conditions with complex causes, constitutional and degenerative diseases, etc.).

Estimates of the genetic risk per generation are based on a 30-year reproductive generation. That is, the median parental age for production of children is age 30 (one-half the children are produced by persons less than age 30, the other half by persons over age 30). Thus, the radiation dose accumulated up to age 30 is used to estimate the genetic risks. Using this accumulated dose and the number of live births in the population along with the estimated genetic risk per unit dose, it is possible to estimate the total number of genetic effects per year, those in the first generation and the total across all time. Most genetic risk analyses have provided such data. EPA assessment of risks of genetic effects includes both first generation estimates and total genetic burden estimates.

Direct and Indirect Methods of Obtaining Risk Coefficients for Genetic Effects

Genetic effects, as noted above, may occur in the offspring of the exposed individuals or they may be spread across all succeeding generations. Two methods have been used to estimate the frequency of mutations in the offspring of exposed persons, direct and indirect. In either case, the starting point is data from animal studies, not data obtained from studies of human populations.

For a direct estimate, the starting point is the frequency of a mutation per unit exposure in some experimental animal study. The 1982 UNSCEAR report gave an example of the direct method for estimating induction of balanced reciprocal translocations (a type of chromosomal aberration) in males per rad of low level, low-LET radiation (UNSCEAR82). This method required the following six steps:

	Induction rate/rad
(1) Rate of induction in rhesus monkey Spermatogonia: cytogenetic data	0.86×10^{-4}
(2) Rate of induction that relates to recoverable translocations in the F_1 (1st filial generation) progeny [divide (1) by 4]	0.215×10^{-4}
(3) Rate after low dose rate X-rays: based on mouse cytogenetic observations [divide (2) by 2]	0.1075×10^{-4}

(4) Rate after chronic gamma-irradiation: based on mouse cytogenetic observations [divide (2) by 10]		0.022×10^{-4}
(5) Expected rate of unbalanced products: [multiply (3) and (4) by 2]	for (3)	0.215×10^{-4}
	for (4)	0.043×10^{-4}
(6) Expected frequency of congenitally malformed children in the F_1, assuming that about 6 percent of unbalanced products [item (5) above] contribute to this		
	for low dose rate X-rays	1.3×10^{-6}
	for chronic gamma radiation	$\approx 0.3 \times 10^{-6}$

For humans, UNSCEAR estimates that as a consequence of induction of balanced reciprocal translocations in exposed fathers, an estimated 0.3 to 1.3 congenitally malformed children would occur in each 10^6 live births for every rad of parental radiation exposure.

A complete direct estimate of genetic effects would include estimates, derived in a manner similar to that shown above for each type of genetic damage. These direct estimates can be used to calculate the risk of genetic effects in the first generation (F_1) children of exposed parents.

The indirect (or doubling dose) method of estimating genetic risk also uses animal data but in a different way. The 1980 BEIR-3 report demonstrates how such estimates are obtained (NAS80).

(1) Average radiation-induced mutation per gene for both sexes in mice [based on 12 locus data in male mice]: induction rate per rad	0.25×10^{-7}
(2) Estimated human spontaneous mutation rate per gene	0.5×10^{-6} to 0.5×10^{-5}
(3) Relative mutation risk in humans [divide (1) by (2)]	0.005 to 0.05
(4) Doubling dose: the exposure needed to double the human mutation rate	20 to 200 rad

The doubling dose can then be used to estimate the equilibrium genetic effects or the genetic burden in all future generations caused by the exposure of parents. Since the genetic component of congenital defects occurring in the population can be estimated by epidemiological surveys, and this component is considered to be maintained at an equilibrium level by mutations, a doubling dose of ionizing radiation would double these genetic effects. Dividing the number of the various genetic effects in 10^6 live-births by the doubling dose yields the estimate of genetic effects per rad. For example:

(1)	Autosomal dominant and x-linked diseases, current incidence	10,000 per 10^6 live births
(2)	Estimated doubling dose	20 to 200 rad
(3)	Estimate of induced autosomal dominant and x-linked diseases	50 to 500 per 10^6 live births per rad of parental exposure

A doubling dose estimate assumes that the total population of both sexes is equally irradiated, as occurs from background radiation, and that the population exposed is large enough so that all genetic damage can be expressed in future offspring. Although it is basically an estimate of the total genetic burden across all future generations, it can also provide an estimate of effects that occur in the first generation. Usually a fraction of the total genetic burden for each type of damage is assigned to the first generation using population genetics data as a basis to determine the fraction. For example, the BEIR-3 committee geneticists estimated that one-sixth of the total genetic burden of x-linked mutations would be expressed in the first generation, five-sixths across all future generations. EPA assessment of risks of genetic effects includes both first generation estimates and total genetic burden estimates.

6.5.2 Estimates of Genetic Harm Resulting from Low-LET Radiations

One of the first estimates of genetic risk was made in 1956 by the NAS Committee on the Biological Effects of Atomic Radiation (BEAR Committee). Based on Drosophila (fruit fly) data and other considerations, the BEAR Genetics Committee estimated that 10 Roentgens (10 R*) per generation continued indefinitely would lead to about 5,000 new instances of "tangible inherited defects" per 10^6 births, and about one-tenth of them would occur in the first generation after the irradiation began (NAS72). The UNSCEAR addressed genetic risk in their 1958, 1962, and 1966 reports (UNSCEAR58,62,66). During this period, they estimated one rad of low-LET radiation would cause a 1 to 10 percent increase in the spontaneous incidence of genetic effects.

In 1972, both the NAS BEIR Committee and UNSCEAR reexamined the question of genetic risks (NAS72, UNSCEAR72). Although there were no definitive human data, additional information was available on the genetic effects of radiation on mammals and insects. In 1977, UNSCEAR reevaluated the 1972 genetics estimates (UNSCEAR77). Their new estimates used recent information on the current incidence of various genetic conditions, along with additional data on radiation exposure of mice and marmosets and other considerations.

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*R is the symbol for Roentgen, a unit of measurement of x-radiation, equivalent to an absorbed dose in tissue of approximately 0.9 rad.

In 1980, an ICRP Task Group (ICRPTG) summarized recommendations that formed the basis for the genetic risk estimates published in ICRP Report 26 (Of80). These risk estimates provided in Table 6.5-1, are based on data similar to that used by the BEIR and UNSCEAR Committees, but with slightly different assumptions and effect categories.

Table 6.5-1. ICRP task group estimate of number of cases of serious genetic ill health in liveborn from parents irradiated with 10^6 man-rem in a population of constant size(a) (Assumed doubling dose = 100 rad)

Category of genetic effect	First generation	Equilibrium
Unbalanced translocations: risk of malformed liveborn	23	30
Trisomics and XO	30	30
Simple dominants and sex-linked mutations	20	100
Dominants of incomplete penetrance and multifactorial disease maintained by mutation	16	160
Multifactorial disease not maintained by mutation	0	0
Recessive disease	--	--
Total	89	320

(a)This is equivalent to effects per 10^6 liveborn following an average parental population exposure of 1 rem per 30-year generation, as used by BEIR and UNSCEAR.

Source: (Of80).

In 1980 the NAS BEIR-3 Committee revised the genetic risk estimates (NAS80). The revision considered much of the same material that was in BEIR-1, the newer material considered by UNSCEAR in 1977, and some additional data. Estimates for the first generation are about a factor of two smaller than reported in the BEIR-1 report. For all generations, the new estimates are essentially the same as shown in Table 6.5-2.

Table 6.5-2. BEIR-3 estimates of genetic effects of an average population exposure of 1 rem per 30-year generation

Type of genetic disorder	Current incidence per 10^6 liveborn	Effects per 10^6 liveborn per rem per generation	
			First generation
Equilibrium			
Autosomal dominant and x-linked	10,000	5-65	40-200
Irregularly inherited	90,000	(not estimated)	20-900
Recessive	1,100	Very few	Very slow increase
Chromosomal aberrations	6,000	Fewer than 10	Increases only slightly
Total	107,100	5-75	60-1100

Source: (NAS80).

The most recent genetic risk estimates are given in Table 6.5-3 and include some new data on cells in culture and the results of genetic experiments using primates rather than rodents (UNSCEAR82).

Although all of the reports described above used somewhat different sources of information, there is reasonable agreement in the estimates presented in Table 6.5-4. Most of the difference is caused by the newer information used in each report. Note that all estimates listed above are based on the extrapolation of animal data to humans. Groups differ in their interpretation of how genetic experiments in animals might be expressed in humans. While there are no comparable human data at present, information on hereditary defects among the children of A-bomb survivors provides a degree of confidence that the animal data do not lead to underestimates of the genetic risk following exposure to humans. (See "Observations on Human Populations" which follows.)

It should be noted that the genetic risk estimates summarized in Table 6.5-4 are for low-LET, low dose, and low dose rate irradiation. Much of the data were obtained from high dose rate studies, and most authors have used a sex-averaged factor of 0.3 to correct for the change from high dose rate, low-LET to low dose rate, low-LET exposure (NAS72,80, UNSCEAR72,77). However, factors of 0.5 to 0.1 have also been used in estimates of specific types of genetic damage (UNSCEAR72,77,82).

Table 6.5-3. UNSCEAR 1982 estimated effect of 1 rad per generation of low dose or low dose rate, low-LET radiation on a population of 10^6 liveborn according to the doubling dose method (Assumed doubling dose = 100 rad)

Disease classification	Current incidence	Effect of 1 rad per generation	
		First generation	Equilibrium
Autosomal dominant and x-linked diseases	10,000	15	100
Recessive diseases	2,500	Slight	slow increase
Chromosomal diseases			
Structural	400	2.4	4
Numerical	3,000	Probably very small	
Congenital anomalies, anomalies expressed later, constitutional and degenerative diseases	90,000	4.5	45
Total	105,900	22	149

Source: (UNSCEAR82).

6.5.3 Estimates of Genetic Harm for High-LET Radiations

Although genetic risk estimates are made for low-LET radiation, some radioactive elements, deposited in the ovary or testis can irradiate the germ cells with alpha particles. The ratio of the dose (rad) of low-LET radiation to the dose of high-LET radiation producing the same endpoint is called RBE and is a measure of the effectiveness of high-LET compared to low-LET radiation in causing the same specific endpoint.

Studies with the beta particle emitting isotopes carbon-14 and tritium yielded RBE's of 1.0 and 0.7 to about 2.0, respectively (UNSCEAR82). At the present time, the RBE for genetic endpoints due to beta particles is taken as one (UNSCEAR77,82).

Studies of the RBE for alpha-emitting elements in germinal tissue have used only plutonium-239. Studies comparing cytogenetic endpoints after chronic low dose rate gammma radiation exposure, or incorporation of plutonium-239 in the mouse testis, have yielded RBE's of 23 to 50 for the type of genetic injury (reciprocal translocations) that might be transmitted to liveborn offspring (NAS80, UNSCEAR77,82). However, an RBE of four for plutonium-239 compared to chronic low-LET radiation was reported for specific locus mutations observed in neonate mice (NAS80). Neutron RBE, determined from cytogenetic studies in mice, also ranges from about four to 50 (UNSCEAR82, Gr83a, Ga82). Most reports use an RBE of 20 to convert risk estimates for low dose rate, low-LET radiation to risk estimates for high-LET radiation.

Table 6.5-4. Summary of genetic risk estimates per 10^6 liveborn for an average population exposure of 1 rad of low dose or low dose rate, low-LET radiation in a 30-year generation

Source	Serious hereditary effects	
	First generation	Equilibrium (all generations)
BEAR, 1956 (NAS72)	---	500
BEIR-I, 1972 (NAS72)	49[a] (12-200)[b]	300[a] (60-1500)
UNSCEAR, 1972 (UNSCEAR72)	9[a] (6-15)	300
UNSCEAR, 1977 (UNSCEAR77)	63	185
ICRPTG, 1980 (Of80)	89	320
BEIR-3, 1980 (NAS80)	19[a] (5-75)	257[a] (60-1100)
UNSCEAR, 1982 (UNSCEAR82)	22	149

[a] Geometric mean is calculated by taking the square root of the product of two numbers for which the mean is to be calculated. The cube root of three numbers, etc. In general, it is the N^{th} root of the product of N numbers for which the mean is to be calculated.

[b] Numbers in parentheses are the range of estimates.

6.5.4 Uncertainty in Estimates of Radiogenetic Harm

Chromosomal damage and mutations have been demonstrated in cells in culture, in plants, in insects, and in mammals (UNSCEAR72,77,82). Chromosome studies in peripheral blood lymphocytes of persons exposed to radiation have shown a dose-related increase in chromosome aberrations (structural damage to chromosome) (UNSCEAR82). In a study of nuclear dockyard workers exposed to external x-radiation at rates of less than five rads per year, Evans, et al. found a significant increase in the incidence of chromosome aberrations (Ev79). The increase appeared to have a linear dependence on cumulative dose. In a study of people working and living in a high natural background area where there was both external gamma-radiation and internal alpha-radiation, Pohl-Ruling et al. reported a complex dose response curve (Po78). For mainly gamma-radiation exposure (less than 10 percent alpha radiation), they reported that the increase in chromosome aberrations increased linearly from 100 to 200 mrads per year then plateaued from 300 mrads to 2 rads per year. They concluded:

> "From these data, and data in the literature, it can be concluded that the initial part of the dose-effect curve for chromosome aberrations is not linear or sigmoid with a threshold at the lowest dose, but rises sharply and passes into a complex upward form with a kind of plateau until it meets the linear curve of the high dose."

Although chromosomal damage in peripheral blood lymphocytes cannot be used for predicting genetic risk in progeny of exposed persons, it is believed by some to be a direct expression of the damage, analogous to that induced in germ cells, resulting from the radiation exposure. It is at least evidence that chromosome damage can occur *in vivo* in humans.

Since there is no quantitative human data on genetic risks following radiation exposure, risk estimates are based on extrapolations from animal data. As genetic studies proceeded, emphasis has shifted from *Drosophila* to mammalian species in attempts to find an experimental system which would reasonably project what might happen in humans.

For example, Van Buul reported the slope (b) of the linear regression, Y = a + bD, for induction of reciprocal translocations in spermatogonia (one of the stages of sperm development) in various species as follows (Va80):

Specie	$b \times 10^4 \pm sd \times 10^4$
Rhesus monkey	0.86 ± 0.04
Mouse	1.29 ± 0.02 to 2.90 ± 0.34
Rabbit	1.48 ± 0.13
Guinea pig	0.91 ± 0.10
Marmoset	7.44 ± 0.95
Human	3.40 ± 0.72

These data indicate that animal-based estimates for this type of genetic effect would be within a factor of four of the true human value. In this case most of the animal results would underestimate the risk in humans.

However, when risk estimates such as this are used in direct estimation of risk for the first generation, the total uncertainty in the estimate becomes indeterminate. Even if studies have been made in a species which can predict the dose response and risk coefficient for a specific radiation-induced genetic damage, there is no certainty that it species, are used to adjust the risk coefficient to what is expected for humans. The uncertainty in these extrapolations has not been quantified.

A rough estimate of the uncertainty can be obtained by comparing direct estimates of risk for the first generation with doubling-dose estimates in the 1977 UNSCEAR report (UNSCEAR77). The estimates differ by a factor between two and six with the direct estimate usually smaller than the doubling dose estimate.

A basic assumption in the doubling dose method of estimation is that there is a proportionality between radiation-induced and spontaneous mutation rates. Some of the uncertainty was removed in the 1982 UNSCEAR report with the observation that in two test systems (fruit flies and bacteria), there is a proportionality between spontaneous and induced mutation rates at a number of individual gene sites. There is still some question as to whether the sites that have been examined are representative of all sites and all gene loci or not. The doubling dose estimated dose, however, seems better supported than the direct estimate.

While there is still some uncertainty as to what should be doubled, future studies on genetic conditions and diseases can only increase the total number of such conditions. Every report, from the 1972 NAS and UNSCEAR reports to the most recent, has listed an increased number of conditions and diseases which have a genetic component.

Observations on Human Populations

As noted earlier, the genetic risk estimates are based on interpretation of animal experiments as applied to data on naturally-occurring hereditary diseases and defects in man. A study of birth cohorts was initiated in the Japanese A-bomb survivors in mid-1946. This resulted in a detailed monograph by Neel and Schull which outlined the background of the first study and made a detailed analysis of the findings to January 1954 when the study terminated (Ne56). The authors concluded only that it was improbable that human genes were so sensitive that exposures as low as 3 R, or even 10 R, would double the mutation rate. While this first study addressed morphological endpoints, subsequent studies have addressed other endpoints. The most recent reports on this birth cohort of 70,082 persons have attempted only to estimate the minimum doubling dose for genetic effects in humans (Sc81, Sa82).

Data on four endpoints have been recorded for this birth cohort. Frequency of stillbirths, major congenital defects, prenatal death, and frequency of death prior to age 17 have been examined in the entire cohort. Frequency of cytogenetic aberrations (sex chromosome aneuploidy) and frequency of biochemical variants (a variant enzyme or protein electrophoresis pattern) have been measured on large subsets of this cohort.

Although the updated data reported appear to suggest radiation effects have occurred, the numbers are small and not statistically significant. Overall, the estimated doubling dose for low-LET radiation at high doses and dose rates for human genetic effects is about 156 rem and 250 rem (Sc81, Sa82). As noted above, animal studies indicate that chronic exposures to low-LET radiation would be less hazardous by a factor of three (NAS72,80). This would increase the estimated doubling dose to 468 rem to 750 rem, respectively. These recent reports suggest the minimum doubling dose for humans may be four to seven times higher than those in Table 6.5-4 (based on animal data). It would be premature to reach a firm decision on the exact amount since these reports are based on the T65 dosimetry in Japan which is being revised. However, we believe EPA estimates of genetic risks will prove to be conservative even when the dosimetry of A-bomb survivors is revised.

EPA is using the geometric mean of the BEIR-3 range of doubling doses, about 110 rads. The minimum doubling dose reported above is four to seven times greater. It is unlikely that dose estimates for Japanese survivors will change by this much (RERF83,84). Therefore, EPA believes the estimate of doubling of about 100 rads will continue to be a conservative estimate.

Ranges of Estimates Provided by Various Models

EPA has continued to follow the recommendations of the 1980 BEIR-3 committee and uses a linear nonthreshold model for estimating genetic effects. Although, as pointed out by the 1982 UNSCEAR committee, there are a number of models other than linear ($Y = c + ad$), e.g., linear quadratic ($Y = c + bD + eD^2$), quadratic ($Y = k + fD^2$), even power function ($Y = k + gD^h$). However, there are strong data to support the hypothesis that mutations themselves are single track events. That is, the mutations follow a linear dose response function while the observed mutation rate shows the influence of other factors, and may be nonlinear (UNSCEAR82). Y is yield of genetic effects; D is radiation dose; c, C, k, and K are spontaneous incidence constants for genetic effects; and a, b, e, f, g, and h are the rate constants for radiation induced genetic effects.

Most of the arguments for a nonlinear dose response have been based on target theory (Le62) or microdosimetric site theory (Ke72). However, other theories based on biology [e.g., enzyme induction-saturation (Go80,82), repair-misrepair (To80)] could also provide models that fit the observed data. There is still much disagreement on which dose response model is appropriate for estimating genetic effects in humans. Until there is more consensus, the linear nonthreshold model appears to be a prudent approach that will not grossly underestimate the risks.

The agreement in estimates made on a linear nonthreshold model in various reports is reasonably good. Even though the authors of the reports used different animal models, interpreted them in different ways, and had different estimates of the level of human genetic conditions in the population, the range of risk coefficients is about an order of magnitude (see Table 6.5-4). For the most recent, more comparable estimates, the range is a factor of two to four (see ICRPTG, BEIR-3 and UNSCEAR82 in Table 6.5-4).

6.5.5 The EPA Genetic Risk Estimate

There is no compelling evidence for preferring any one set of the genetic risk estimates listed in Table 6.5-4. EPA has used the estimates from BEIR-3 (NAS80). These "indirect" estimates are calculated using the normal prevalence of genetic defects and the dose that is considered to double this risk. The NAS estimates which EPA uses are based on a "doubling dose" range with a lower bound of 50 rems and an upper bound of 250 rems. We prefer these risk estimates to those made by the ICRP task group, which used a "direct" estimate because the ICRPTG tabulation combines "direct" estimates for some types of genetic damage with doubling dose estimates for others (Of80). We also prefer the BEIR-3 risk estimates to the "direct" estimates of UNSCEAR82 which tabulates genetic risk separately by the direct method and by the doubling dose method. The risk estimated by the direct method does not include the same types of damage estimated by doubling doses and was not considered further. Moreover, the BEIR-3 genetic risk estimates provide a better estimate of uncertainty than the UNSCEAR82 and ICRPTG estimates because the BEIR-3 Committee assigned a range of uncertainty for multifactorial diseases (>5 percent to <50 percent) which reflects the uncertainty in the numbers better than the other estimates do (5 percent and 10 percent, respectively).

In developing the average mutation rate for the two sexes used in the calculation of the relative mutation risk, the BEIR-3 Committee postulated that the induced mutation rate in females was about 40 percent of that in males (NAS80). Recent studies by Dobson et al. suggest that the assumption was invalid and that human oocytes should have a risk equivalent to that of human spermatogonia. This would increase the risk estimate obtained from doubling dose methods by a factor of 1.43 (Do83a, Do83b, Do84a, Do84b).

We recognize, however, that the use of the doubling dose concept does assume that radiation-induced genetic damage is in some way proportional to "spontaneous" damage. As noted earlier, the recent evidence obtained in insects (Drosophila) and bacteria (E. coli) supports the hypothesis that, with the exception of "hot spots" for mutation, the radiation-induced mutation rate is proportional to the spontaneous rate (UNSCEAR82). No proof that this is also true in mammals is available yet.

The BEIR-3 estimates give a considerable range. To express the range as a single estimate, the geometric mean of the range is used, a method first recommended by UNSCEAR for purposes of calculating genetic risk (UNSCEAR58). The factor of three increase in risk for high dose rate, low-LET radiation noted earlier is also used.

The question of RBE for high-LET radiation is more difficult. As noted above, estimated RBE's for plutonium-239 alphas versus chronic gamma radiation for reciprocal translocations as determined by cytogenetic analyses are between 23 and 50 (NAS80, UNSCEAR82). However, the observed RBE for single locus mutations in developing offspring of male mice given plutonium-239 compared to those given X-ray irradiation is four (NAS80).

The average of RBE's for reciprocal translocations and for specific locus mutations is 20.25. Since reported neutron RBE's are similar to those listed above for plutonium-239 alpha radiation, we use an RBE of 20 to estimate genetic risks for all high-LET radiations. This is consistent with the RBE for high-LET particles recommended for estimated genetic risks associated with space flight (Gr83b).

Genetic risk estimates used by EPA for high- and low-LET radiations are listed in Table 6.5-5. As noted above, EPA uses the dose received before age 30 in assessing genetic risks.

Table 6.5-5. Estimated frequency of genetic disorders in a birth cohort due to exposure of the parents to 1 rad per generation

Radiation	Serious Heritable Disorders (Cases per 10^6 liveborn)			
	First Generation		All Generations	
	low[(a)]	high[(b)]	low[(a)]	high[(b)]
Low Dose Rate, Low-LET	20	30	260	370
High Dose Rate, Low-LET	60	90	780	1110
High-LET	400	600	5200	7400

(a) Female sensitivity to induction of genetic effects is 40 percent as great as that of males.

(b) Female sensitivity to induction of genetic effects is equal to that of males.

The EPA estimates in Table 6.5-5 are limited, like all other human genetic risk estimates, by the lack of confirming evidence of genetic effects in humans. These estimates depend on a presumed resemblance of radiation effects in animals to those in humans. The magnitude of the possible error is indeterminable. The study with the largest data base, the Japanese A-bomb survivors, appears, at best, to provide only an estimate of the minimum doubling dose for calculating the maximum genetic risk in man. However, doubling dose estimates are also uncertain since the number of human disorders having a recognized genetic component is constantly increasing, and the type of genetic damage implicated in a specific disorder may change. The combined uncertainties in doubling dose estimates and the magnitude of genetic contributions to various disorders probably introduce an overall uncertainty of about an order of magnitude in the risk estimates. Moreover, the BEIR Committee in deriving its estimate has assumed that almost all of the risk was due to recessive mutations which would eventually be eliminated. To what extent this occurs will depend on medical practices in the future. It is possible, as our knowledge of medicine improves, that recessive hereditary defects will be carried on for many more generations than assumed by the BEIR Committee.

The relative risk of high-LET radiation compared to low dose rate, low-LET radiation (RBE) is also uncertain. The data are sparse, and different studies often used different endpoints. In addition, the microscopic dosimetry, i.e., the actual absorbed dose in the cells at risk, is poorly known. However, the RBE estimate used by EPA should be within a factor of five of the true RBE for high-LET radiation.

6.5.6 Teratogenic Effects

Although human teratogenesis (congenital abnormalities or defects) associated with x-ray exposure has a long history, the early literature deals mostly with case reports. Stettner reported a case in 1921 and Murphy and Goldstein studied a series of pregnancies in which 18 of the children born to 76 irradiated mothers were microcephalic (St21, Mu29, Go29). However, the irradiation exposures were high.

In 1930, Murphy exposed some rats to X-rays at doses of 200 R to 1600 R. Thirty-four of 120 exposed females had litters, and five of the litters had animals with developmental defects (Mu30). He felt that this study confirmed his clinical observations and earlier reports of animal studies. Although there were additional studies of radiation-induced mammalian teratogenesis before 1950, the majority of the studies were done after that time (see Ru53 for a review), perhaps reflecting radiation hazards caused by the explosion of nuclear weapons in 1945 (Ja70).

Much of the work done after World War II was done in mice and rats (Ru50,54,56, Wi54, Hi54). Early studies, at relatively high radiation exposures, 25 R and above, established some dose response relationships. More importantly, they established the time table of sensitivity of the developing rodent embryo and fetus to radiation effects (Ru54, Hi53, Se69, Hi66).

Rugh, in his review of radiation teratogenesis listed the reported mammalian anomalies and the exposure causing them (Ru70). The lowest reported exposure was 12.5 R for structural defects and 1 R for functional defects. He also suggested human exposure between ovulation and about 7 weeks gestational age could lead to structural defects and from about 6 weeks gestational age until birth could lead to functional defects. In a later review, he suggested structural defects in the skeleton might be induced as late as the 10th week of gestation and functional defects as early as the 4th week (Ru71). It should be noted that the gestation period in mice is much shorter than that in humans and that weeks of gestation referred to above are in terms of equivalent stages of mouse-human development. Estimates of equivalent gestational age are not very accurate.

In the reports of animal studies it appeared as if teratologic effects, other than perhaps growth retardation, had a threshold for induction of effects (Ru54,53, Wi54). However, Ohzu showed that doses as low as 5 R to preimplantation mouse embryos caused increased resorption of implanted embryos and structural abnormalities in survivors (Oh65). Then in 1970, Jacobsen reported a study in which mice were exposed to 5, 20 or 100 R on the 8th day of pregnancy (Ja70). He concluded that the dose response function for induction of skeletal effects was linear, or nearly linear, with no observable threshold. This appears consistent with a report by Russell, which suggested a threshold for some effects whereas others appeared linear (Ru57).

Rugh suggested there may be no threshold for radiation-induced congenital effects in the early human fetus (Ru71). In the case of microcephaly and mental retardation, at least this may be the case. For other teratogenic effects, the dose response in humans is unknown. In 1978, Michel and Fritz-Niggli reported induction of a significant increase in growth retardation, eye and nervous system abnormalities, and post implantation losses in mice exposed to 1 R (Mi78). The increase was still greater if there was concurrent exposure to radiosensitizing chemicals such as iodoacetimide or tetracycline (Mi78).

One of the problems with the teratologic studies in animals is the difficulty of determining how dose response data should be interpreted.

Russell pointed out some aspects of the problem: 1) although radiation is absorbed throughout the embryo, it causes selective damage which is consistently dependent on the stage of embryonic development at the time of irradiation, and 2) the damaged parts respond, in a consistent manner, within a narrow time range (Ru54). However, while low dose irradiation at a certain stage of development produces changes only in components at their peak sensitivity, higher doses may induce additional abnormalities which have peak sensitivity at other stages of development, and may further modify expression of the changes induced in parts of the embryo at peak sensitivity during the time of irradiation. In the first case, damage may be to primordial cells themselves, while in the second, the damage may lead indirectly to the same or different endpoints.

The embryo/fetus starts as a single fertilized egg and divides and differentiates to produce the normal infant at term. (The embryonic period, when organs develop, is the period from conception to 7 weeks gestational age. The fetal period, a time of in utero growth, is the period from 8 weeks gestational age to birth.) The different organ and tissue primordia develop independently and at different rates. However, they are in contact through chemical induction or evaporation (Ar54). These chemical messages between cells are important in bringing about orderly development and the correct timing and fitting together of parts of organs or organisms. While radiation can disrupt this pattern, interpretation of the response may be difficult. Since the cells in the embryo/fetus differentiate, divide, and proliferate at different times during gestation and at different rates, gestational times when cells of specific organs or tissues reach maximum sensitivity to radiation are different. Each embryo/fetus has a different timetable. In fact, each half (left/right) of an embryo/fetus may have a slightly different timetable.

In addition, there is a continuum of variation from the hypothetical normal to the extreme deviant, which is obviously recognizable. There is no logical place to draw a line of separation between normal and abnormal. The distinction between minor variations of normal and frank malformation, therefore, is an arbitrary one, and each investigator must establish his own criteria and apply them to spontaneous and induced abnormalities alike (HWC73). For example, some classify mental retardation based on IQ (80 or lower), some classify based on ability to converse or hold a job, some on the basis of the need to be institutionalized.

Because of the problems in interpretation listed above, it appears a pragmatic approach is useful. The dose response should be given as the simplest function that fits the data, often linear or linear with a threshold. No attempt should be made to develop complex dose response models unless the evidence is unequivocal.

The first report of congenital abnormalities in children exposed in utero to radiation from atomic bombs was that of Plummer (P152). Twelve children with microcephaly of which 10 also had mental retardation had been identified in Hiroshima in the in utero exposed survivors. They were found as part of a program started in 1950 to study children exposed in the first trimester of gestation. In 1955 the program was expanded to include all survivors exposed in utero.

Studies initiated during the program have shown the following radiation-related effects: 1) growth retardation; 2) increased microcephaly; 3) increased mortality, especially infant mortality; 4) temporary suppression of antibody production against influenza; and 5) increased frequency of chromosomal aberrations in peripheral lymphocytes (Ka73).

Although there have been a number of studies of Japanese A-bomb survivors, including one showing a dose and gestational age related increase in postnatal mortality (Ka73), only incidence of microcephaly and mental retardation have been investigated to any great extent. In the most recent report, Otake and Schull showed that mental retardation was associated with exposure between 8 and 15 weeks of gestation (10 to 17 weeks of gestation if counted from the last menstrual period) (Ot83). They further found a linear dose response relationship for induction of mental retardation that had a slope yielding a doubling dose for mental retardation of about 2 rads, fetal absorbed dose (Ot83). Classification as mentally retarded was based on "unable to perform simple calculations, to care for himself or herself, or if he or she was completely unmanageable or had been institutionalized" (Ot83).

Estimates of the risk of mental retardation for a rad of embryo/fetus exposure in the U.S. population can be derived by three methods. The first and easiest method is to use the absolute risk calculated by Otake and Schull for the Japanese survivors (Ot84). A second method is to use the doubling dose calculated by Otake and Schull times the incidence of mental retardation per 10^3 live births (Ot83).

Unfortunately, a number of assumptions must be made to establish the incidence of mental retardation per 10^3 live births. Mental retardation may be classified as mild (IQ 50-70), moderate (IQ 35-49), severe (IQ 20-34) and profound (IQ <20) (WHO75). However, some investigators use only mild mental retardation (IQ 50-70) and severe mental retardation (IQ <50) as classes (Ha81, St84). Mental retardation is not usually diagnosed at birth but at some later time, often at school age. Since the mental retardation may have been caused before or during gestation, at the time of birth, or at some time after birth, that fraction caused before or during gestation must be estimated. In like manner since mental retardation caused before birth may be due to genetic conditions, infections, physiologic conditions, etc., the fraction related to unknown causes during gestation must be estimated. This is the fraction that might possibly be doubled by radiation exposure.

A third method to estimate the risk is indirectly using the relationship of microcephaly and mental retardation reported in the Japanese survivors (Wo65, Ot83). If head size is assumed to be normally distributed, then the fraction of the population with a head size 2 or 3 standard deviations smaller than average can be obtained from statistical tables. The fraction of 10^3 liveborn with microcephaly multiplied by the proportion of mental retardation associated with that head size yields an estimate of the incidence of mental retardation per 10^3 live births, which can then be used with the doubling dose to estimate the risk as described above.

Risk estimates for mental retardation are derived below for comparison purposes using each of the three methods described above.

Estimate of Incidence Per Rad Based on Direct Application of the Slope of the Japanese Data

Otake and Schull gave an estimate of "The Relationship of Mental Retardation to Absorbed Fetal Exposure in the 'Sensitive' Period When All 'Controls' are Combined" (Ot84). The estimate of 0.416 cases of mental retardation per 100 rad could be directly applicable to a U.S. population. In this case the risk estimate would be about 4 cases of mental retardation per rad per 1000 live births.

Estimate of Incidence Per Rad Based on the Doubling Dose

The Otake and Schull report suggested the doubling dose for mental retardation was about 2 rads fetal absorbed dose or about a 50 percent increase in mental retardation per rad (Ot83). It would seem reasonable that this doubling dose would apply only to ideopathic cases of mental retardation caused during gestation, that is, those which have no known genetic, viral, bacterial, etc., cause.

Data from studies of the prevalence of mental retardation in school age populations in developed countries suggest a prevalence of 2.8 cases/1000 (Uppsala County, Sweden) to 7.4 cases/1000 (Amsterdam, Holland) of severe mental retardation, with a mean of about 4.3 ± 1.3 cases/1000 (St84). Where data are available for males and females separately, the male rate is about 30 percent higher than the female rate (St84). Historically, the prevalence of mild mental retardation has been 6 to 10 times greater than that of severe mental retardation. But in recent Swedish studies, the rates of prevalence of mild and severe mental retardation have been similar (St84). This was suggested to be due to a decline in the "cultural-familial syndrome". That is, improved nutrition, decline in infection and diseases of childhood, increased social and intellectual stimulation, etc., combined to reduce the proportion of nonorganic mental retardation and, therefore, the prevalence of mild mental retardation (St84).

In studies of the causes of mental retardation, 23 to 42 percent of the mental retardation has no identified cause (Gu77, Ha81, St84). It is this portion of the mental retardation which may be susceptible to increase from radiation exposure of the embryo/fetus. In that case, the prevalence of ideopathic mental retardation would be 0.6 to 3.1 cases per 1000 of severe mental retardation and perhaps an equal number of cases of mild mental retardation.

For purposes of estimating the effects of radiation exposure of the embryo/fetus, a risk of spontaneous ideopathic mental retardation of 1 to 6 per 1000 will be used. If this spontaneous ideopathic mental retardation can be increased by radiation the estimate would be:

(1 to 6 cases per 1000 live births)(0.5 increase per rad)

or about 0.5 to 3 cases of mental retardation per rad per 1000 live births.

This estimate may be biased low because mental retardation induced during gestation is often associated with high childhood death rate (St84). If this is generally true for ideopathic causes of mental retardation, it would cause an underestimation of the risk.

Estimate of Incidence Per Rad Based on Incidence of Microcephaly

1) Of live born children, 2.275 percent will have a head circumference 2 standard deviations or more smaller than average, 0.621 percent will have a head circumference 2.5 standard deviations or more smaller than average, and 0.135 percent will have a head circumference 3 standard deviations or more smaller than average (statistical estimate based on a normal distribution).

2) There is evidence in a nonselected group of 9,379 children that mental retardation can be estimated using incidence of microcephaly, even though head circumference in the absence of other supporting data, e.g., height or proportion, is an uncertain indicator of mental retardation. Based on a study of 9,379 children, Nelson and Deutschberger concluded that about half of the children with a head circumference 2.5 standard deviations or more smaller than average had IQ's of 79 or lower

(Ne70). Since 0.67 percent of those studied were in this group, the observed number is about what would be expected based on the normal distribution of head size in a population, 0.62 percent. The estimated incidence of mental retardation per live birth in a population would be:

$$\frac{\text{6.7 cases of microcephaly}}{\text{1000 live births}} \times \frac{\text{0.5 cases of mental retardation}}{\text{case of microcephaly}}$$

or about 3.4 cases of mental retardation per 1000 live births.

3) A first approximation of risk of mental retardation might then be:

$$\frac{\text{3.4 cases of mental retardation}}{\text{1000 live births}} \times \frac{\text{0.5 increase}}{\text{rad}}$$

or about 2 cases of mental retardation per 1000 live births per rad.

Both microcephaly and mental retardation were increased in Japanese survivors (Wo65,66). About half of those with head sizes 2 or more standard deviations smaller than average had mental retardation (RERF78), a result similar to that observed by Nelson and Deutschberger (Ne70). Therefore, the above estimate based on the incidence of microcephaly in a population should be a reasonable estimate of the risk from radiation.

Summary of the Calculated Risk of Mental Retardation

The risk of increased mental retardation per rad of embryo/fetus exposure during the 8- to 15-week gestational period estimated above ranges from about 5×10^{-4} to 4×10^{-3} cases per live birth, the larger being a direct estimate. The geometric mean of these estimates is 1.4×10^{-3}; the arithmetic mean is 2.4×10^{-3} cases per live birth.

All the estimates derived above by any of the three methods are in the same range as an earlier UNSCEAR estimate of an increase of 1×10^{-3} cases of mental retardation per rad per live birth (UNSCEAR77). The UNSCEAR estimate, however, did not consider gestational age at the time of exposure. The Otake and Schull report did address gestational age and estimated a higher risk, but a narrower window of susceptibility (Ot83).

If the estimates are applicable, the 15 mrads of low-LET background radiation delivered during the 8- to 15-week gestational age-sensitive period could induce a risk of 6×10^{-5} to 7.5×10^{-6} cases of mental retardation per live birth. This can be compared to an estimate of a spontaneous occurrence of 1.5×10^{-2} to 3.4×10^{-3} cases of mental retardation per live birth.

Japanese A-bomb survivors exposed in utero also showed a number of structural abnormalities and, particularly in those who were microcephalic, retarded growth (Wo65). No estimate has been made of the radiation-related incidence or dose response relationships for these abnormalities.

However, UNSCEAR made a very tentative estimate based on animal studies that the increased incidence of structural abnormalities in animals may be 5 x 10^{-3} cases per R per live birth, but stated that projection to humans was unwarranted (UNSCEAR77). In any event, the available human data cannot show whether the risk estimates derived from high dose animal data overestimates the risk in humans.

It should be noted that all of the above estimates are based on high dose rate low-LET exposure. UNSCEAR in 1977 also investigated the dose rate question and stated:

> "In conclusion, the majority of the data available for most species indicate a decrease of the cellular and malformature effects by lowering the dose rate or by fractionating the dose. However, deviations from this trend have been well documented in a few instances and are not inconsistent with the knowledge about mechanisms of the teratogenic effects. It is therefore impossible to assume that dose rate and fractionation factors have the same influence on all teratological effects." (UNSCEAR77).

From this analysis, EPA has concluded that a range of risk is 4 x 10^{-3} to 5 x 10^{-4} cases of mental retardation per live birth per rad of low-LET radiation delivered between weeks 8 and 15 of gestation with no threshold identified at this time.

No attempt can be made now to estimate total teratogenic effects. However, it should be noted that the 1977 UNSCEAR estimate from animals was 5 x 10^{-3} cases of structural abnormalities per R per live birth (about the same number per rad of low-LET). This estimate must be viewed as a minimum one since it is based, to a large extent, on observation of grossly visible malformations. Differences in criteria for identifying malformations have compounded the problem, and questions of threshold and species differences have made risk projection to humans unwarranted.

6.5.7 Nonstochastic Effects

Nonstochastic effects, those effects that increase in severity with increasing dose and may have a threshold, have been reviewed in the 1982 UNSCEAR report (UNSCEAR82). In general, acute doses of 10 rads of low-LET radiation and higher are required to induce these effects. It is possible that some of the observed effects of in utero exposure are nonstochastic, e.g., the risk of embryonic loss, estimated to be 10^{-2} per R (UNSCEAR77), following radiation exposure soon after fertilization. However, there are no data to address the question. Usually, no nonstochastic effects of radiation are expected at environmental levels of radiation exposure.

6.6 Radiation Risk - A Perspective

To provide a perspective on the risk of fatal radiogenic cancers and the hereditary damage due to radiation, we have calculated the risk from background radiation to the U.S. population using the risk coefficients presented in this chapter and the computer codes described in Appendix A. The risk resulting from background radiation is a useful perspective for the risks caused by releases of radionuclides. Unlike cigarette smoking, auto accidents, and other measures of common risks, the risks resulting from background radiation are neither voluntary nor the result of alcohol abuse. The risk caused by background radiation is very largely unavoidable; therefore, it is a good benchmark for judging the estimated risks from radionuclide releases. Moreover, to the degree that the estimated risk of radionuclides is biased, the same bias is present in the risk estimates for background radiation.

Low-LET background radiation has three major components: cosmic radiation, which averages to about 28 mrads per year in the U.S.; terrestrial sources, such as radium in soil, which contributes an average of 26 mrads per year (NCRP75); and the low-LET dose resulting from internal emitters. The last differs between organs, to some extent, but for soft tissues is about 24 mrads per year (NCRP75). Fallout from nuclear weapons tests, naturally occurring radioactive materials in buildings, etc., contribute about another 10 mrems for a total low-LET whole-body dose of about 90 mrads per year. The lung and bone receive somewhat larger doses due to high-LET radiations; see below. Although extremes do occur, the distribution of this background annual dose to the U.S. population is relatively narrow. A population weighted analysis indicates that 80 percent of the U.S. population would receive annual doses that are between 75 mrads per year and 115 mrads per year (EPA81).

As outlined in Section 6.2, the BEIR-3 linear models yield, for lifetime exposure to low-LET radiation, an average lifetime risk of fatal radiogenic cancer of 280 per 10^6 person rad. Note that this average is for a group having the age- and sex-specific mortality rates of the 1970 U.S. population. We can use this datum to calculate the average lifetime risk due to low-LET background radiation as follows. The average duration of exposure in this group is 70.7 years and at 9×10^{-2} rad per year, the average lifetime dose is 6.36 rads. The risk of fatal cancer per person in this group is:

$$\frac{280 \text{ fatalities}}{10^6 \text{ person rad}} \times 6.36 \text{ rads} = 1.78 \times 10^{-3}$$

or about 0.18 percent of all deaths. The vital statistics we use in our radiation risk analyses indicate that the probability of dying from cancer in the United States from all causes is about 0.16, i.e., 16 percent. Thus, the 0.18 percent result for the BEIR-3 linear dose response model indicates that about 1 percent of all U.S. cancer is due to low-LET background radiation. The BEIR-3 linear quadratic model indicates that about 0.07 percent of all deaths are due to low-LET background radiation or about 0.4 percent of all cancer deaths.

Table 6.3-1 indicates a risk of 460 fatalities per 10^6 organ rad for alpha emitters in lung tissue. The lifetime cancer from this exposure is:

$$\frac{460 \text{ fatalities}}{10^6 \text{ organ rad}} \times \frac{0.03 \text{ rad}}{\text{year}} \times 70.7 \text{ years} = 0.98 \times 10^{-3}$$

This is twice the risk due to low-LET background radiation calculated by means of the BEIR-3 linear quadratic model and more than half of the risk calculated by means of the BEIR-3 linear model.

The 1982 UNSCEAR report indicates that the average annual dose to the endosteal surfaces of bone due to naturally-occurring high-LET alpha radiation is about 6 mrads per year or, for a quality factor 20, 120 mrems per year (UNSCEAR82). Table 6.3-1 indicates that the lifetime risk of fatal bone cancer due to this portion of the naturally occurring radiation background is:

$$\frac{20 \text{ cases}}{10^6 \text{ person rad}} \times \frac{0.006 \text{ rad}}{\text{year}} \times 70.7 \text{ years} = 8.5 \times 10^{-6}$$

The spontaneous incidence of serious congenital and genetic abnormalities has been estimated to be about 105,000 per 10^6 live births, about 10.5 percent of live births (NAS80, UNSCEAR82). The low-LET background radiation dose of about 90 mrads/year in soft tissue results in a genetically significant dose of 2.7 rads during the 30-year reproductive generation. Since this dose would have occurred in a large number of generations, the genetic effects of the radiation exposure are thought to be an equilibrium level of expression. Since genetic risk estimates vary by a factor of 20 or more, EPA uses a log mean of this range to obtain an average value for estimating genetic risk. Based on this average value, the background radiation causes 700 to 1000 genetic effects per 10^6 live births, depending on whether or not the oocyte is as sensitive to radiation as the spermatogonia. This result indicates that about 0.67 percent to 0.95 percent of the current spontaneous incidence of serious congenital and genetic abnormalities may be due to the low-LET background radiation.

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Chapter 7: MOVEMENT AND HEALTH RISKS OF RADIONUCLIDE RELEASES TO THE ACCESSIBLE ENVIRONMENT

7.1 Introduction

This chapter describes analyses used to assess the health risks caused by the environmental transport of radionuclides once they are released from a repository to the accessible environment. As part of its program to develop 40 CFR Part 191, the Agency estimated population health risks for a 10,000-year period following disposal in mined geologic repositories (see Chapter 8). These estimates were used in selecting the containment requirements in the disposal standards. The objective of this chapter is: 1) to describe the environmental pathways that were considered when calculating the environmental risk commitments (ERC's: fatal cancers and serious genetic defects) that could occur as a result of releases of radionuclides from disposal systems; and 2) to summarize the results of these calculations. A complete description of the analysis is described in Sm85. This chapter also describes how the release limits of the containment requirements were derived from the results of these calculations.

In performing these long-term assessments of population health effects, the Agency recognizes that it is pointless to try to make precise projections of the actual risks due to radionuclide releases from repositories. Population distributions, food chains, living habits, and technological capabilities will undoubtedly change in major ways over 10,000 years. Unlike geological processes, they can be realistically predicted only for relatively short times. Accordingly, very general models of environmental pathways were formulated as opposed to the detailed analytical techniques that would be appropriate for near-term environmental assessments of specific facilities. Population characteristics similar to those of today were assumed.

The models discussed in this chapter consider risks to populations, as opposed to risks to individuals. Therefore, individual risks caused by potential releases from a repository cannot be determined from these analyses. Analyses that assess individual risks are described in Chapter 8.

7.2 Methodology

Radionuclides can be released from geologic repositories and move through the environment through four pathways: 1) to surface water (e.g., a river) through ground water, 2) to an ocean through surface water, 3) to a land surface directly, or 4) to multiple pathways after the very unlikely possibility of disruption by a volcano or a meteorite. For each of these four release modes, radionuclide movement through the geosphere and the biosphere to the population was modeled, and an estimate was made of the intake by or exposure to the population through each of these environmental pathways. The environmental pathways included for each of the release modes are described in Table 7.2-1.

Risk conversion factors per unit intake or per unit external exposure were applied to the radionuclide concentrations output by the model to estimate fatal cancers and serious genetic effects to all generations per curie of each different radionuclide released to the accessible environment. The results were used to specify the release limits in Table 1 of 40 CFR Part 191, based on a consideration of only excess fatal cancers. The genetic effects to all generations were lower than the estimated fatal cancers by a factor of two or more for all radionuclides and were not used to establish the release limits. The risk conversion factors used to estimate fatal cancers are listed in Table 7.2-2 (Sm85).

Health effects were calculated for the entire population exposed to the releases from a repository; calculations were not terminated at some arbitrary distance from the repository. A time integration was performed to obtain the sum of the health effects from the time the repository is sealed ("disposal") until a specified time in the future (usually 10,000 years after disposal). The radioactivity intakes and exposures were then converted to population ERC's by multiplying by the appropriate risk conversion factors. The following sections summarize the factors considered in the calculation of the population intake of radioactivity for the internal pathways--or the integrated population exposure for the external pathways--for each of the four release modes.

7.3 Releases to Surface Water

In the surface water release pathway, the repository containment is assumed to be breached--after some initial period--and ground water circulates through the repository into the surrounding geologic media and eventually to an aquifer. The aquifer then flows underground until it intersects a river. To determine the total release to the surface water (river), the release rate was integrated over the time period of interest. The integrated release rate, in equation form, was then used to compute surface water concentrations for use with several environmental pathways. These are discussed in the following subsections.

Table 7.2-1. Release modes and environmental pathways

Release mode	Pathways included in this release mode
Releases to river	Drinking water ingestion
	Freshwater fish ingestion
	Food crops ingestion
	Milk ingestion
	Beef ingestion
	Inhalation of resuspended material
	External dose, ground contamination
	External dose, air submersion
Releases to ocean	Ocean fish ingestion
	Ocean shellfish ingestion
Releases directly to land surface	Food crops ingestion
	Milk ingestion
	Beef ingestion
	Inhalation of resuspended material
	External dose, ground contamination
	External dose, air submersion
Releases due to volcano/meteorite interaction	
Releases directly to land	Food crops ingestion
	Milk ingestion
	Beef ingestion
	Inhalation of resuspended material
	External dose, ground contamination
	External dose, air submersion
Releases to air over land	Food crops ingestion
	Milk ingestion
	Beef ingestion
	Inhalation of dispersed and resuspended material
	External dose, ground contamination
	External dose, air submersion
Releases to air over ocean	Ocean fish ingestion
	Ocean shellfish ingestion

Table 7.2-2. Fatal cancer risk conversion factors[a]

Radionuclide	Fatal cancers per Ci intake				Fatal cancers from external doses	
	Inhalation[b]		Ingestion[b]		Air submersion per Ci-y/m^3	Ground contamination per Ci-y/m^2
	1	2	1	2		
C-14	3.05E-3	3.05E-3	4.32E-1	4.32E-1	0	0
Ni-59	4.76E-1	4.76E-1	3.76E-2	3.76E-2	4.10E-2	8.87E-3
Sr-90	4.52E+2	5.19E+1	2.29E+0	2.85E+1	0	0
Zr-93	2.72E+1	6.60E+0	1.27E-1	1.27E-1	1.23E-1	1.89E-2
Tc-99	6.12E+0	6.12E+0	5.37E-1	5.37E-1	5.97E-4	1.41E-5
Sn-126	5.72E+1	5.72E+1	2.04E+0	2.04E+0	2.54E+3	5.11E+1
I-129	1.61E+1	1.61E+1	2.41E+1	2.41E+1	7.57E+0	3.98E-1
Cs-135	1.27E+0	1.27E+0	1.82E+0	1.82E+0	0	0
Cs-137	8.49E+0	8.49E+0	1.24E+1	1.24E+1	7.18E+2	1.43E+1
Sm-151	5.27E+0	5.27E+0	3.46E-2	3.46E-2	8.15E-4	9.43E-5
Pb-210	2.27E+4	2.99E+3	4.13E+2	4.13E+2	1.34E+0	6.09E-2
Ra-226	4.38E+4	5.33E+3	4.91E+2	4.91E+2	2.35E+3	4.20E+1

(continued)

Table 7.2-2. Fatal cancer risk conversion factors[(a)] (continued)

Radionuclide	Fatal cancers per Ci intake				Fatal cancers from external doses	
	Inhalation[(b)]		Ingestion[(b)]		Air submersion per Ci-y/m³	Ground contamination per Ci-y/m²
	1	2	1	2		
Ra-228	7.98E+4	1.58E+4	9.71E+1	9.71E+1	3.43E+3	5.88E+1
Ac-227	6.97E+4	3.74E+4	2.85E+2	2.85E+2	4.81E+2	1.05E+1
Th-229	6.45E+4	2.82E+4	8.55E+1	8.55E+1	3.30E+2	7.46E+0
Th-230	6.89E+4	2.05E+4	5.13E+2	5.13E+2	2.35E+3	4.20E+1
Th-232	1.05E+5	2.94E+4	1.17E+2	1.17E+2	3.43E+3	5.88E+1
Pa-231	1.03E+5	6.19E+4	4.67E+2	4.67E+2	5.17E+2	1.14E+1
U-233	2.42E+4	3.70E+3	5.21E+0	5.07E+1	1.68E+1	3.84E-1
U-234	2.07E+4	2.26E+3	9.38E-1	4.61E+1	1.63E-1	1.63E-2
U-235	2.03E+4	2.72E+3	5.86E+0	5.02E+1	2.01+2	4.60E+0
U-236	1.96E+4	2.14E+3	8.86E-1	4.35E+1	1.27E-1	1.48E-2
U-238	1.86E+4	2.04E+3	1.99E+0	4.84E+1	2.36E+1	5.17E-1
Np-237	2.89E+4	2.46E+4	1.86E+2	1.86E+2	2.83E+2	6.39E+0

(continued)

Table 7.2-2. Fatal cancer risk conversion factors[a] (continued)

Radionuclide	Fatal cancers per Ci intake				Fatal cancers from external doses	
	Inhalation[b]		Ingestion[b]		Air submersion per Ci-y/m^3	Ground contamination per Ci-y/m^2
	1	2	1	2		
Pu-238	3.13E+4	2.49E+4	1.86E+2	1.86E+2	8.80E-2	1.68E-2
Pu-239	3.09E+4	2.65E+4	2.04E+1	2.00E+2	9.06E-2	7.64E-3
Pu-240	3.09E+4	2.65E+4	2.04E+1	1.99E+2	8.63E-2	1.61E-2
Pu-241	1.19E+4	1.23E+3	9.57E+0	9.57E+0	5.98E-1	1.87E-2
Pu-242	2.94E+4	2.52E+4	1.94E+1	1.90E+2	7.36E-2	1.34E-2
Am-241	3.27E+4	2.75E+4	2.07E+2	2.07E+22	1.99E+1	6.24E-1
Am-243	3.19E+4	2.73E+4	2.06E+2	2.06E+2	2.55E+2	5.95E+0
Cm-245	6.54E+4	5.58E+4	4.21E+2	4.21E+2	1.02E+2	2.58E+0
Cm-246	3.26E+4	2.78E+4	2.10E+2	2.10E+2	6.81E-2	1.41E-2

(a) The fatal cancer risk conversion factors in this table are the sum of the risk factors for the listed radionuclide plus any significant daughter products which can develop during the residence time of the radionuclide in the accessible environment.

(b) When a radionuclide can exist having more than one solubility class, factor 1 refers to the form with a lower solubility class, and factor 2 refers to the form with a higher solubility class (Sm85).

Source: Sm85

7.3.1 Drinking Water

It was assumed that the population receives 65 percent of its drinking water from surface waters with no reduction in radionuclide concentrations due to water treatment (Mu77). The annual intake rate of drinking water and water-based drinks by an individual is 600 liters (ICRP75); thus, 390 liters was assumed to be supplied by surface water. The average ratio of the population drinking water to the river flow rate is 3.3 x 10^{-7} person-year/liter based upon an assumed world population of 10 billion persons and an annual flow rate for the world's rivers of 3 x 10^{16} liters (UNSCEAR77). This ratio is within the range of similar values associated with various river basins in the United States (Sm85). The average fraction of a river's flow that is used for drinking water is obtained by combining the fraction of drinking water which is surface water, the drinking water rate for an individual, and the average ratio of population drinking water to the river flow rate. This is numerically the same as the total intake of a radionuclide by the population per curie of radionuclide released to the surface water.

7.3.2 Ingestion of Fish

Fish caught in the river are assumed to contain radionuclides due to uptake from the water. The amount of radionuclides accumulated in the fish (in terms of Ci per kg of fish body weight) is a direct function of the radionuclide concentration (Ci per liter) in the surface water. The fraction of the radionuclides released to the surface water that is ingested by the population through fish consumption is obtained by calculating the quantity of radionuclides in the fish through the use of bioaccumulation factors, and by determining an average ratio of the population's fish ingestion rate to the river flow rate (3.3 x 10^{-7} man-kilogram/liter*). The bioaccumlation factors for fish are given in Table 7.3-1.

7.3.3 Ingestion of Food Raised on Irrigated Land

Surface water containing radionuclides released from the repository may be used to spray or irrigate farm land, leading to direct deposition of radionuclides onto the crops and the land surface below the crops. The average fraction of the river flow used for irrigation was assumed to be 0.1, based on the United States average of 0.07 (Sm85, Mu77). In addition, irrigated plants that had incorporated radionuclides through their leaves and root systems are consumed by humans as food, or are consumed by either dairy or beef cattle that transfer radionuclides to milk and meat. The amounts of radioactivity consumed through these pathways was determined by using a radionuclide-specific intake factor for each pathway (food crops, milk, and beef) as given in Table 7.3-2, the fraction of the river flow used for irrigation, and the average number of people that can be fed per unit area of land by each of the pathways as given in Table 7.3-3 (Sm85). The average consumption of

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*This ratio is determined by multiplying the person-year/liter (discussed in Section 7.3.1) by the assumed annual individual fish consumption of 1.0 kg/year (UNSCEAR77).

Table 7.3-1. Bioaccumulation factors for freshwater fish

Radionuclide	Bioaccumulation factor (Ci/kg per Ci/liter)
C-14	NA[a]
Ni-59	1.00E+2
Sr-90	1.10E+1 (Ho79)
Zr-93	3.33E+0
Tc-99	4.30E+1 (B182)
Sn-126	3.00E+3
I-129	3.30E+1 (Ho79)
Cs-135	1.30E+3 (Ho79)
Cs-137	1.30E+3 (Ho79)
Sm-151	2.50E+1
Pb-210	1.00E+2
Ra-226	5.00E+1
Ra-228	5.00E+1
Ac-227	2.50E+1
Th-229	3.00E+1
Th-230	3.00E+1
Th-232	3.00E+1
Pa-231	1.10E+1
U-233	1.00E+1
U-234	1.00E+1
U-235	1.00E+1
U-236	1.00E+1
U-238	1.00E+1
Np-237	5.00E+2 (Sc83)
Pu-238	8.00E+0 (Ri83)
Pu-239	8.00E+0 (Ri83)
Pu-240	8.00E+0 (Ri83)
Pu-241	8.00E+0 (Ri83)
Pu-242	8.00E+0 (Ri83)
Am-241	8.10E+1 (Ri83)
Am-243	8.10E+1 (Ri83)
Cm-245	2.50E+1
Cm-246	2.50E+1

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[a] NA - Not Applicable.

Source: Th72 (unless otherwise noted)

Table 7.3-2. Radionuclide intake factors for farm products raised in areas using contaminated irrigation water

	Radionuclide intake factor (Ci intake per Ci/m^2 deposited)		
Radionuclide	Food Crops	Milk	Meat
C-14	NA[(a)]	NA	NA
Ni-59	4.38E+0	3.22E-1	2.48E-1
Sr-90	2.57E+0	1.07E+0	8.20E-2
Zr-93	4.21E+0	8.18E-2	2.10E+1
Tc-99	1.57E+0	4.00E+0	1.31E+0
Sn-126	1.10E+0	3.04E-1	9.36E+0
I-129	1.17E+1	1.03E+1	2.78E+0
Cs-135	1.40E+1	8.04E+0	8.84E+0
Cs-137	8.51E-1	1.74E+0	1.91E+0
Sm-151	5.47E-1	4.54E-3	4.37E-1
Pb-210	4.98E-1	5.75E-2	2.66E-2
Ra-226	6.62E-1	1.26E-1	6.26E-2
Ra-228	3.95E-1	9.81E-2	4.53E-2
Ac-227	3.95E-1	4.36E-3	2.10E-3
Th-229	7.33E-1	1.49E-3	6.87E-4
Th-230	2.77E+0	3.87E-3	1.79E-3
Th-232	6.73E+0	8.51E-3	3.93E-3
Pa-231	6.92E-1	1.43E-3	1.10E-3
U-233	1.19E+0	1.57E-1	2.01E-2
U-234	1.19E+0	1.57E-1	2.01E-2
U-235	1.19E+0	1.57E-1	2.01E-2
U-236	1.19E+0	1.57E-1	2.01E-2
U-238	1.19E+0	1.57E-1	2.01E-2
Np-237	5.42E-1	2.52E-3	1.94E-2
Pu-238	3.92E-1	2.17E-5	1.67E-4
Pu-239	4.77E-1	2.37E-5	1.83E-4
Pu-240	4.53E-1	2.32E-5	1.79E-4
Pu-241	3.90E-1	2.17E-5	1.67E-4
Pu-242	4.89E-1	2.40E-5	1.85E-4
Am-241	4.35E-1	9.45E-5	3.18E-4
Am-243	4.87E-1	1.03E-4	3.48E-4
Cm-245	4.10E-1	4.67E-3	3.14E-4
Cm-246	4.08E-1	4.63E-3	3.12E-4

(a) NA - Not Applicable.

Source: Sm85

these various food products was then determined. Combining the consumption of food products with the radionuclide content of the products yields an estimate of the fraction of the radionuclides released to the surface water that are transferred to crops by irrigation and ultimately consumed by populations.

Table 7.3-3. Values for persons fed per unit area of land

Food	Person fed/m^2
Vegetative Food Crops	4.79 E-3
Milk	1.56 E-3
Meat	7.85 E-5

Source: Sm85

7.3.4 Inhalation of Resuspended Material

Some of the radionuclides deposited on the soil by irrigation are resuspended into the air. The air concentration of resuspended radionuclides corresponding to the fraction of radionuclides released to the surface water that wind up in water used for irrigation is calculated using a resuspension factor of 10^{-9}/m and the integrated soil surface concentration (Be76, Ne78). The population intake of these radionuclides is then calculated using an annual inhalation rate of 8400 cubic meters and an average population density of 6.7×10^{-5} persons per square meter (ICRP75, UNSCEAR77, Wo79).

7.3.5 External Exposure from Air Submersion

The radionuclides resuspended into the air can also cause submersion exposures to the population. These exposures are also based on the integrated air concentration to which the population is exposed and are calculated from the integrated air concentration, the average population density, and a shielding and occupancy factor 0.33 (UNSCEAR77, Wo79).

7.3.6 External Exposure from Ground Contamination

Finally, the radionuclides deposited on the ground during irrigation can also cause external exposures to persons in the area. Throughout the irrigation period, radionuclides continue to build up on the ground until either irrigation stops or equilibrium is reached with losses through the soil. The methods for estimating these exposures are similar to those applied for air submersion.

7.4 Releases to an Ocean

Releases to a surface water system are assumed to subsequently discharge into an ocean. Since radionuclide decay during travel in the river or depletion of the radionuclide inventory due to river water use and sedimentation is not considered, the radionuclide releases to an ocean are equal to the releases to a surface water. The ocean pathway model has two compartments consisting of a shallow upper layer in which it is assumed that all edible seafood is grown, and a lower layer that includes the remainder of the ocean. Differential equations were developed whose solutions describe the quantities of radionuclides in these two compartments over time. The equation for the upper compartment inventory was divided by the volume of the compartment to determine the time-dependent concentation of radionuclides in the upper layer. This concentration was then used to estimate the fraction of the radionuclides released to the river that is consumed by the population due to bioaccumulation of radionuclides in ocean fish and shellfish.

7.5 Releases Directly to Land Surface

For the land surface pathway models, some of the radioactive waste from the repository is assumed to be brought to the surface after an event such as inadvertent intrusion while drilling for resources. Such releases to the surface are assumed to be over a small area and a short period of time; as such, they can be modeled as instantaneous point sources. The mechanisms distributing the material to humans are resuspension and subsequent dispersion in the atmosphere. After the initial release to the land surface is determined, a time-dependent release rate to the air is estimated using a simple exponential model that depletes the land surface source to account for resuspension and radioactive decay. This release rate is applied in conjunction with an atmospheric dispersion equation to predict air concentrations as a function of time and distance from the source; these air concentrations are then used to estimate ground surface concentrations as a function of time and distance. Once ground surface concentrations are determined, the techniques used to calculate population intake are similar to those described for the surface water release mode. The pathways considered for releases to land surface are ingestion of food raised on land contaminated with radionuclides, including food crops, milk, and meat; inhalation of resuspended radionuclides; external exposure due to air submersion; and external exposure due to ground contamination.

7.6 Releases Due to a Volcanic Eruption or Meteorite Impact

Releases to the land surface and directly to the air can be caused by the extremely unlikely events of disruption by volcanoes or meteorites. The methodology described for the land surface release mode is used for the material released to the land surface. For the material released to the air, it is assumed that the radionuclides would be quickly dispersed in such a manner that they would eventually be distributed uniformly within the troposphere. The airborne material is divided into the fraction over land and the fraction over water using the ratio of earth land surface and earth water surface. Compartment models, with their systems of coupled differential equations, were used to estimate the quantity of radionuclides reaching the land surface or ocean. Finally, the amount of radionuclides or radiation exposure reaching people was estimated through the same pathways described for the land surface or the ocean, respectively.

7.7 Special Considerations for Carbon-14 Environmental Risk Commitment

Unlike the other radionuclides considered in these analyses, stable carbon constitutes a significant fraction of the elemental composition of the human body and man's diet. Thus, transport processes through the different environmental pathways and within plants, animals, and man that apply to trace quantities of other radionuclides do not necessarily apply to radionuclides such as carbon-14 (C-14), where the corresponding stable elements are present in such quantities that saturation effects are significant (Mo79).

Atmospheric releases of C-14 as carbon dioxide can be evaluated using a diffusion-type model of the carbon cycle developed by Killough (Ki77). It seems clear that this model is the correct calculational procedure to use for releases for the volcano/meteorite release mode where it is assumed that high temperatures would cause carbon releases to be oxidized to carbon dioxide. Models are not available to explicitly treat the ERC calculations for C-14 released to water, land surfaces, or air in a chemical form other than carbon dioxide. A review of the literature indicated that the chemical form of C-14 released in the water and land surface release modes is not well known. Also, the rate of oxidation to carbon dioxide of other chemical forms of C-14 over the extensive integration period is not known for these release modes. Considering all these uncertainties, it was concluded that the most prudent course was to use the Killough carbon dioxide model for all four release modes, realizing that this probably leads to conservative estimates of the ERC for the water and land release modes.

The environmental risk commitment for C-14 is obtained by calculating the total body environmental dose commitment (EDC) and multiplying by a fatal cancer risk conversion factor. Values of the total body environmental dose commitment per curie of C-14 released to the atmosphere have been calculated by Fowler using the Killough model (Fo79, Ki77). It is estimated that the ingestion pathway contributes 99 percent of the carbon-14 environmental dose commitment (Fo76); however, it is assumed that the ingestion pathways contribute 100 percent for purposes of computational convenience. For estimating the environmental dose commitment, Fowler's curve of worldwide EDC to the total body per curie release versus time after release was used.

The environmental risk commitment is obtained by multiplying the total body environmental dose commitment by the fatal cancer risk factor of 1.46 x 10^{-4} fatal cancers per total body man-rem as given by Fowler[*] (Fo79). For C-14, this is the total environmental risk commitment for all the pathways within each release mode; the methodology is not applied separately for each pathway.

7.8 Fatal Cancers per Curie Released to the Accessible Environment

This section presents the results of all analyses in terms of the premature fatal cancers induced (over 10,000 years) for each curie of the various radionuclides that may be released to the accessible environment. These fatal cancer estimates have been used to develop the radionuclide release limits in Table 1 of 40 CFR Part 191 of the final rule. The fatal cancer estimates for releases to surface water (the sum of releases to a river and releases to the ocean), to land surfaces, and to the atmosphere are tabulated in Table 7.8-1. Table 7.8-2 shows how the various environmental pathways contribute to the fatal cancer per curie released estimate for releases to surface water. As can be seen from Table 7.8-2, the dominant pathway for each radionuclide is usually ingestion of surface crops irrigated with contaminated water.

7.8.1 Development of Release Limits for 40 CFR Part 191

The analyses described in this chapter were used to develop radionuclide release limits that correspond to the level of protection chosen for the containment requirements of the final rule (Section 191.13). Since releases to surface water through ground water are usually the most important release mode for mined repositories, and since the health effects per curie released are usually the highest for this release mode, the release limits in 40 CFR Part 191 were based solely on the surface water release mode.

To develop the release limits, the appropriate population risk level must first be chosen. The Agency has chosen to base the containment requirements on a population risk level of no more than 1,000 premature cancer deaths over 10,000 years from disposal of 100,000 metric tons of heavy metal (MTHM) contained in spent fuel (or from disposal of the high-level radioactive wastes produced by this much spent fuel). For convenience, the release limits in 40 CFR Part 191 are stated in terms of 1,000 MTHM and can be adjusted to reflect the actual amount of waste in a disposal system. Therefore, the release limits in 40 CFR Part 191 are to be the amount of each radionuclide that would cause 10 health effects over 10,000 years.

Table 7.8-3 summarizes the procedure used to arrive at the release limits in 40 CFR Part 191, Table 1 of the final rule. First, the number of fatal cancers caused per curie released to surface water for each radionuclide (the first column of Tables 7.8-1 and 7.8-2) was divided

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[*] This C-14 fatal cancer risk factor is less than that used for most other radionuclides because a large percentage of the total body dose from C-14 is to adipose tissue and is not effective in producing cancer (Fo79).

Table 7.8-1. Fatal cancers per curie released to the accessible environment for different release modes

Radionuclide	Releases to surface water	Releases to land surface	Releases due to violent interactions(a)
C-14	5.83E-02	5.83E-02	5.83E-02
Ni-59	4.78E-05	6.79E-07	2.89E-05
Sr-90	2.26E-02	3.76E-05	1.16E-03
Zr-93	1.59E-04	2.26E-05	1.22E-04
Tc-99	3.68E-04	5.65E-08	1.99E-04
Sn-126	1.25E-02	1.38E-03	2.73E-02
I-129	8.09E-02	3.96E-03	5.57E-02
Cs-135	7.76E-03	5.75E-04	4.91E-03
Cs-137	1.07E-02	2.19E-05	3.39E-03
Sm-151	9.78E-06	6.71E-08	4.72E-06
Pb-210	1.25E-01	1.52E-04	4.31E-02
Ra-226	1.68E-01	5.62E-03	7.20E-02
Ra-228	2.42E-02	1.57E-05	2.78E-02
Ac-227	6.87E-02	1.24E-04	3.82E-02
Th-229	6.20E-02	1.90E-02	5.06E-02
Th-230	7.25E-01	3.86E-01	1.26E+00
Th-232	3.83E-01	3.76E-01	3.73E-01
Pa-231	1.50E-01	2.36E-02	1.28E-01
U-233	2.18E-02	7.51E-04	7.75E-03
U-234	1.98E-02	6.54E-04	5.94E-03
U-235	2.19E-02	8.42E-04	8.27E-03
U-236	1.87E-02	6.18E-04	5.62E-03
U-238	2.08E-02	6.90E-04	5.67E-03
Np-237	8.66E-02	1.21E-04	2.83E-02
Pu-238	4.27E-02	3.10E-04	2.07E-02
Pu-239	5.20E-02	6.23E-03	1.20E-02
Pu-240	5.03E-02	5.22E-03	1.15E-02
Pu-241	2.18E-03	2.50E-06	9.36E-04
Pu-242	5.01E-02	6.34E-03	1.09E-02
Am-241	5.80E-02	1.05E-03	2.54E-02
Am-243	6.81E-02	2.45E-03	3.40E-02
Cm-245	1.24E-01	8.08E-03	6.09E-02
Cm-246	6.00E-02	3.54E-03	2.89E-02

(a) Interactions of a metorite or a volcanic eruption with a repository.

Table 7.8-2. Fatal cancers per curie released to the accessible environment for releases to surface water

Radio-nuclide	TOTAL	Ingestion							Inhalation	External dose	
		Drinking water	Freshwater fish	Surface crops	Milk	Beef	Ocean fish	Ocean shellfish	Resuspended material	Ground contamination	Air submersion
C - 14	5.83E-02	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Ni- 59	4.78E-05	4.91E-06	1.25E-06	3.94E-05	4.72E-07	1.83E-08	1.20E-06	5.00E-07	3.25E-10	3.17E-10	1.11E-15
Sr- 90	2.26E-02	3.72E-03	1.04E-04	1.75E-02	1.19E-03	4.59E-06	2.30E-06	3.84E-06	4.05E-09	0.00E+00	0.00E+00
Zr- 93	1.59E-04	1.66E-05	1.41E-07	1.28E-04	4.05E-07	5.23E-06	8.05E-06	5.37E-07	6.58E-08	1.45E-07	4.86E-14
Tc- 99	3.68E-04	7.02E-05	7.70E-06	2.02E-04	8.38E-05	1.38E-06	1.73E-06	1.44E-06	4.67E-11	0.00E+00	1.80E-19
Sn-126	1.25E-02	2.67E-04	2.04E-03	5.37E-04	2.42E-05	3.75E-05	1.96E-03	1.09E-04	6.47E-08	7.55E-03	1.14E-10
I -129	8.09E-02	3.15E-03	2.65E-04	6.75E-02	9.68E-03	1.31E-04	7.82E-05	6.15E-05	3.68E-08	5.41E-06	6.86E-13
Cs-135	7.76E-03	2.38E-04	7.89E-04	6.10E-03	5.71E-04	3.16E-05	2.36E-05	2.46E-06	5.38E-09	0.00E+00	0.00E+00
Cs-137	1.07E-02	1.62E-03	5.37E-03	2.53E-03	8.42E-04	4.65E-05	2.07E-06	2.15E-06	1.33E-09	3.19E-04	4.45E-12
Sm-151	9.78E-06	4.52E-06	2.88E-07	4.53E-06	6.13E-09	2.97E-08	5.23E-08	3.49E-07	2.14E-09	0.00E+00	1.31E-17
Pb-210	1.25E-01	5.40E-02	1.38E-02	4.93E-02	9.26E-04	2.16E-05	4.42E-03	2.46E-03	3.45E-07	9.60E-08	6.13E-15
Ra-226	1.68E-01	6.41E-02	8.18E-03	7.78E-02	2.41E-03	6.03E-05	4.05E-03	1.35E-03	8.91E-06	1.00E-02	1.56E-10
Ra-228	2.42E-02	1.27E-02	1.62E-03	9.19E-03	3.71E-04	8.63E-06	8.02E-05	2.67E-05	5.61E-07	2.23E-04	4.83E-12
Ac-227	6.87E-02	3.72E-02	2.37E-03	2.70E-02	4.85E-05	1.17E-06	2.53E-04	1.69E-03	4.29E-06	1.07E-04	2.18E-12
Th-229	6.20E-02	1.12E-02	8.55E-04	1.50E-02	4.97E-06	1.15E-07	2.03E-02	6.76E-03	4.85E-04	7.39E-03	2.25E-10
Th-230	7.25E-01	6.70E-02	5.13E-03	3.40E-01	7.74E-05	1.80E-06	1.40E-01	4.67E-02	4.29E-04	1.25E-01	1.95E-09
Th-232	3.83E-01	1.53E-02	1.17E-03	1.89E-01	3.88E-05	9.02E-07	3.24E-02	1.08E-02	6.27E-04	1.34E-01	2.90E-09
Pa-231	1.50E-01	6.10E-02	1.71E-03	7.74E-02	2.60E-05	1.01E-06	1.43E-03	2.38E-04	5.33E-04	7.58E-03	1.76E-10
U -233	2.18E-02	6.62E-03	1.69E-04	1.44E-02	3.10E-04	2.00E-06	1.63E-04	2.71E-05	7.41E-06	3.43E-05	1.33E-12
U -234	1.98E-02	6.02E-03	1.54E-04	1.31E-02	2.82E-04	1.82E-06	1.48E-04	2.47E-05	4.53E-06	5.63E-07	1.29E-14
U -235	2.19E-02	6.56E-03	1.67E-04	1.43E-02	3.07E-04	1.98E-06	1.62E-04	2.70E-05	5.46E-06	4.00E-04	1.60E-11
U -236	1.87E-02	5.68E-03	1.45E-04	1.24E-02	2.66E-04	1.72E-06	1.41E-04	2.34E-05	4.29E-06	4.41E-09	1.01E-14
U -238	2.08E-02	6.32E-03	1.61E-04	1.38E-02	2.96E-04	1.91E-06	1.57E-04	2.61E-05	4.09E-06	2.65E-05	1.88E-12
Np-237	8.66E-02	2.43E-02	3.10E-02	2.41E-02	1.83E-05	7.08E-06	6.03E-03	1.00E-03	3.40E-06	4.83E-05	1.55E-12
Pu-238	4.27E-02	2.43E-02	4.96E-04	1.75E-02	1.57E-07	6.10E-08	3.35E-05	3.73E-04	1.14E-05	1.74E-09	1.60E-15
Pu-239	5.20E-02	2.61E-02	5.33E-04	2.28E-02	1.85E-07	7.18E-08	1.81E-04	2.01E-03	3.14E-04	2.21E-08	4.26E-14
Pu-240	5.03E-02	2.60E-02	5.31E-04	2.16E-02	1.80E-07	6.99E-08	1.57E-04	1.75E-03	2.75E-04	3.97E-08	3.55E-14
Pu-241	2.18E-03	1.25E-03	2.55E-05	8.94E-04	8.10E-09	3.14E-09	7.63E-07	8.48E-06	8.73E-08	9.46E-09	1.68E-15
Pu-242	5.01E-02	2.48E-02	5.07E-04	2.23E-02	1.78E-07	6.90E-08	1.80E-04	2.00E-03	3.13E-04	3.95E-08	3.62E-14
Am-241	5.80E-02	2.70E-02	5.59E-03	2.16E-02	7.63E-07	1.29E-07	4.88-04	3.25E-03	3.85E-05	6.22E-06	1.10E-12
Am-243	6.81E-02	2.69E-02	5.56E-03	2.40E-02	8.28E-07	1.41E-07	1.42E-03	9.44E-03	7.92E-05	7.08E-04	2.93E-11
Cm-245	1.24E-01	5.50E-02	3.51E-03	4.13E-02	7.67E-05	2.59E-07	2.98E-03	1.99E-02	3.85E-04	3.49E-04	2.79E-11
Cm-246	6.00E-02	2.74E-02	1.75E-03	2.05E-02	3.79E-05	1.29E-07	1.31E-03	8.75E-0	1.75E-04	2.11E-08	1.70E-14

Table 7.8-3. Development of Release Limits presented in Table 1 of 40 CFR Part 191

Radio-nuclide	Fatal cancers per curie released to surface water(a)	Curies required to cause 10 fatal cancers(b)	Release limit per 1000 MTHM or other unit of waste(c) (curies)
C-14	5.83E-02	172	100
Ni-59(d)	4.78E-05	209,000	1,000(d)
Sr-90	2.26E-02	442	1,000
Zr-93(d)	1.59E-04	62,900	1,000(d)
Tc-99	3.68E-04	27,200	10,000
Sn-126	1.25E-02	800	1,000
I-129	8.09E-02	124	100
Cs-135	7.76E-03	1,290	1,000
Cs-137	1.07E-02	935	1,000
Sm-151(d)	9.78E-06	1,020,000	1,000(d)
Pb-210(e)	1.25E-01	80	100(e)
Ra-226	1.68E-01	60	100
Ra-228(e)	2.42E-02	413	100(e)
Ac-227(e)	6.87E-02	145	100(e)
Th-229(e)	6.20E-02	161	100(e)
Th-230	7.25E-01	14	10
Th-232	3.83E-01	26	10
Pa-231(e)	1.50E-01	67	100(e)
U-233	2.18E-02	459	100
U-234	1.98E-02	505	100
U-235	2.19E-02	457	100
U-236	1.87E-02	535	100
U-238	2.08E-02	480	100
Np-237	8.66E-02	115	100
Pu-238	4.27E-02	234	100
Pu-239	5.20E-02	192	100
Pu-240	5.03E-02	199	100
Pu-241(d)	2.18E-03	4580	1,000(d)
Pu-242	5.01E-02	200	100
Am-241	5.80E-02	172	100
Am-243	6.81E-02	147	100
Cm-245(e)	1.24E-01	81	100(e)
Cm-246(e)	6.00E-02	167	100(e)

(a) From Table 7.8-1.

(b) Equal to 10 fatal cancers ÷ fatal cancers per curie released to surface water (column 2).

(c) Cumulative releases to the accessible environment for 10,000 years after disposal.

(d) Included in 40 CFR Part 191 as any other radionuclide with half-life greater than 20 years that does not emit alpha particle.

(e) Included in 40 CFR Part 191 as any other alpha-emitting radionuclide with a half-life greater than 20 years.

into 10 health effects to determine the number of curies of that radionuclide that would cause 10 health effects (shown in the third column of Table 7.8-3). Then, these estimates were rounded to the nearest order of magnitude to reflect the approximate nature of all of these calculations. (For example, if a number were between 100 and 1,000, it would generally be rounded to 100 if it were less than 316, the logarithmic midpoint, and to 1,000 if it were more than 316). Several judgments were made in this rounding process. First, radionuclides present only in small quantities, or which appear to be insignificant to the overall risk were combined into an "any other radionuclide" category. There are two of these "any other radionuclide" categories, one for alpha-emitting radionuclides and one for non-alpha-emitting radionuclides. Radionuclides included in either of these categories are identified in Table 7.8-3. Each category was assigned a release limit. Also, uncertainties in the long-term risk estimates were considered when rounding values up or down. For example, the projected curies of strontium-90 and the various isotopes of uranium needed to cause 10 health effects are all about the same and are all near the midpoint of the rounding range. However, the release limit for the relatively short-lived strontium-90 was rounded up to 1,000 curies, while the release limits for the very long-lived uranium isotopes (for which ultimate environmental pathways will be more uncertain) were conservatively rounded down to 100 curies.

7.9 Uncertainty Analysis

Environmental pathway dosimetry and risk models generally employ an environmental transport methodology consisting of multiplicative chain algorithms incorporating several variables. When regulatory analyses are performed, the tendency is to choose conservative values for these variables due to the inherent uncertainty in the parameters. The multiplicative nature of the models means that conservatism in chosing values for individual parameters can lead to larger conservatism in the result. The problem with this approach is that widespread conservatism can lead to extremely conservative and sometimes unrealistic results.

The consideration of uncertainty in individual parameter values used in environmental pathway models has been a subject of discussion in the technical community for more than a decade (Ba79a,b, Ho79, Mi79, Ru79, Sh79). However, the comprehensive consideration of overall uncertainty in environmental pathway, dosimetry, and health impact analyses has begun to be addressed only recently (Ri83, Ru83).

When considering the uncertainty in the input parameters associated with environmental pathway calculations, the most common procedure has been to qualitatively consider the range of reported parameter values and to use judgement to select the "best" value to use for a particular application. More recently, attempts have been made to statistically analyze the distribution of data for individual parameters and to choose a mean or median as the "best" value for regulatory purposes.

It appears that the most systematic mechanism for considering uncertainty in multiplicative chain models would be to include a probability distribution representing current uncertainty about parameter values in the input data and to run a sufficient number of cases (with parameter values for each case chosen by a suitable sampling procedure) such that the distribution of results can be evaluated. The results of this type of analysis could be considered in choosing an appropriate set of single-valued parameters to apply for regulatory calculations. Alternately, a decision might be made to perform the regulatory calculations probabilistically, then choose limits for a standard (or to perform calculations to see if a limit is met) at a specified confidence level. We believe the subject needs additional study to determine the most appropriate use of uncertainty analysis for standards setting applications; however, it is clear to us that a quantitative analysis of the uncertainties is most useful for focusing on important uncertainties for more intensive consideration.

Most of the technical analyses discussed in this chapter were performed prior to the increase in emphasis on uncertainty in risk assessment calculations. In most cases, point values for each parameter were chosen after review of the range of values reported in the literature and were chosen to be near the mean or median value to avoid obtaining unrealistically conservative results. Baes has published a very complete review and analysis of parameters used to predict the transport of radionuclides through agricultural pathways (Ba84). For most radionuclides we considered, the surface water release mode was dominant and the food pathways either dominated or were major pathways in determining the fatal cancers per curie released to surface water. Default values from the Baes report were used for many critical food pathway parameters, and Baes states that these default values were chosen to be realistic rather than highly conservative. Since the default values used were based on Baes recent extensive review of the literature, the analysis is more realistic than would be the case if other sources of data were used.

In response to one of the recommendations of the SAB Subcommittee that reviewed the technical basis for 40 CFR Part 191, the Agency asked the Envirosphere Company to perform an uncertainty analysis of the calculations that produced the estimates for fatal cancers per curie of radioactivity released to surface water (Sm85). Envirosphere reviewed the surface water pathway models and identified the key parameters. The uncertainties in these parameters were characterized by probability distributions that were propagated through the models using a simulation technique, which produced uncertainty distributions in the estimates of fatal cancers per unit of radionuclide release to surface water.

An example of the results of the Envirosphere uncertainty analysis is presented in Figure 7.9-1, which shows uncertainty distributions for the fatal cancers risk for Am-243 releases to surface water. Figure 7.9-1 shows a probability plot of the fatal cancers per 10,000 years per curie of Am-243 released to surface water. This figure indicates a 75 percent probability, considering parameter uncertainties, that EPA's release limit of 100 Ci for Am-243 will result in 10 or less deaths over 10,000 years per 1000 MTHM. Similar calculations were performed for several other radionuclides listed in Table 7.8-3 (Sm85, ENV85).

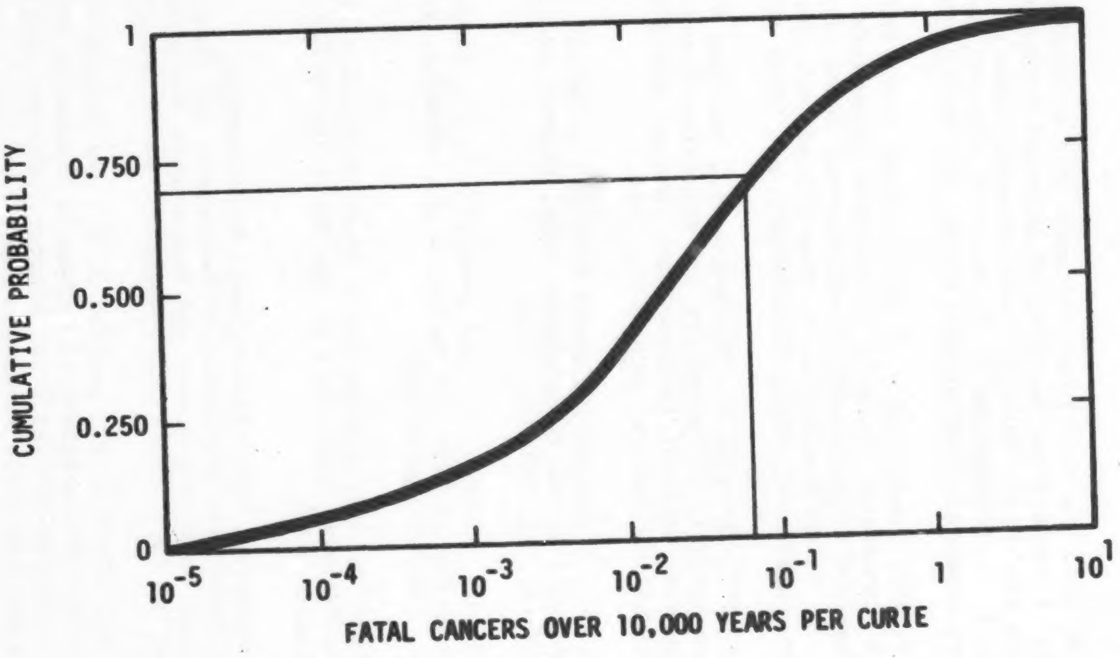

Figure 7.9-1. Probability distribution of population risks per curie of Am-243 released to surface water.

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Chapter 8: RISK ASSESSMENT OF DISPOSAL OF HIGH-LEVEL RADIOACTIVE WASTE IN MINED GEOLOGIC REPOSITORIES

8.1 Introduction

An important consideration in developing the final disposal standards has been the assessment of risks associated with the disposal of these wastes in mined geologic repositories. The risk assessments carried out in support of the development of the standards are intended to be "generic" in nature. In developing the assessments, the Agency considered a wide range of geologic environments and other related parameters. In the early stages of the EPA work, the DOE, which is responsible for developing a geologic repository, had not yet developed extensive data associated with its principal candidate sites. Therefore, the risk assessments conducted in support of the proposed standards were based on the general literature on potential waste disposal environments, as well as the limited data that had been obtained by DOE up to about 1980.

The risk assessments conducted in support of the final rule were improved through the use of additional information available. First, a review of the technical basis of the proposed rule by the Agency's Science Advisory Board (SAB) produced a number of recommendations for revisions to the risk assessments. Many of these SAB recommendations called for the analysis to be less conservative, that is, to use parameters that would tend to produce lower estimates of population risks. In addition, DOE has now developed extensive data on nine specific locations being evaluated for the first repository. These data were available for use in risk assessments for the final rule.

To define the most representative parameters to use in the generic assessments, the Agency has reviewed the developments by DOE at particular sites. The conceptual models were based on these data whenever possible, keeping in mind the recommendations of the SAB. Because of the generic nature of the analysis, the results of the risk calculations cannot purport to project the actual risk expected at these particular sites; such projections will not be possible before the potential sites are fully characterized. Instead, the assessments are intended to provide rough estimates of potential repository performance. Future investigations at a specific site may show that the actual performance at some sites differs significantly from the results of the generic assessments. The Agency, however, has attempted to ensure that its generic calculations are based on the most reasonable parameters and hypothetical scenarios that would control performance at a site.

8.2 Waste Disposal System Model

The waste disposal systems considered in this risk assessment are based on current DOE plans to develop mined geological repositories for disposal of high-level radioactive wastes. Such repositories consist of underground mines or excavations with working levels between 300 and 1000 meters below the surface. The repositories being considered are in rock formations of bedded salt, salt domes, basalt, tuff, and granitic rocks. As described in Section 8.6, this risk assessement examined models of mined geologic repositories in all of these geologic media except for salt domes, which are likely to perform much like bedded salt formations.

The radioactive wastes themselves will consist of either spent fuel from nuclear power reactors or solidified reprocessing wastes in a relatively durable form such as borosilicate glass. These wastes will contain a wide variety of radioactive elements ranging from highly active fission products with relatively short lives to long-lived elements such as transuranic radionuclides created through neutron capture by uranium atoms. For disposal in a mined geologic repository, the wastes would be packaged in canisters that would be placed in holes in the walls or floors of mined rooms in the repository. After emplacement of the wastes, the repository would be backfilled to enhance its mechanical stability and to retard the movement of fluids. The repository's various connections (e.g., shafts and boreholes) to the surface would be severed and sealed. The intent in selecting a repository is to provide a highly stable geologic environment in which ground water contact with the radioactive waste is inhibited or greatly restrained. However, despite the care that will be exercised during the development of a repository, possible future disruptions could lead to the release of radioactive wastes.

The purpose of the risk asessments carried out by the Agency is to identify the most important mechanisms that could lead to releases of radioactive waste to the accessible environment and to estimate the likelihood and consequences of the releases over long time periods in the future. While this analysis varies from one geologic environment to another, the structure of the analysis can be represented as shown in Figure 8.2-1. The components of the system to the right of the vertical dotted line represent the "accessible environment." The components on the left side of the diagram represent the release and transport mechanisms from the repository to the accessible environment.

In order for radioactive wastes to reach the accessible environment, radioactive material must be released from the waste form itself, which might be a borosilicate glass, unprocessed spent fuel, or some similar type of structure. Having left the waste form itself, such radionuclides must escape from the waste canister and then enter the backfilled openings of the repository. Radionuclides may travel from the repository to the accessible environment in two general ways: 1) direct pathways to the land surface, such as might occur if future generations penetrated the repository during an exploratory drilling program and accidentally contacted the wastes, and 2) migration in slowly moving ground water to an aquifer and then to the surface. Details of these environmental pathways were provided in Chapter 7.

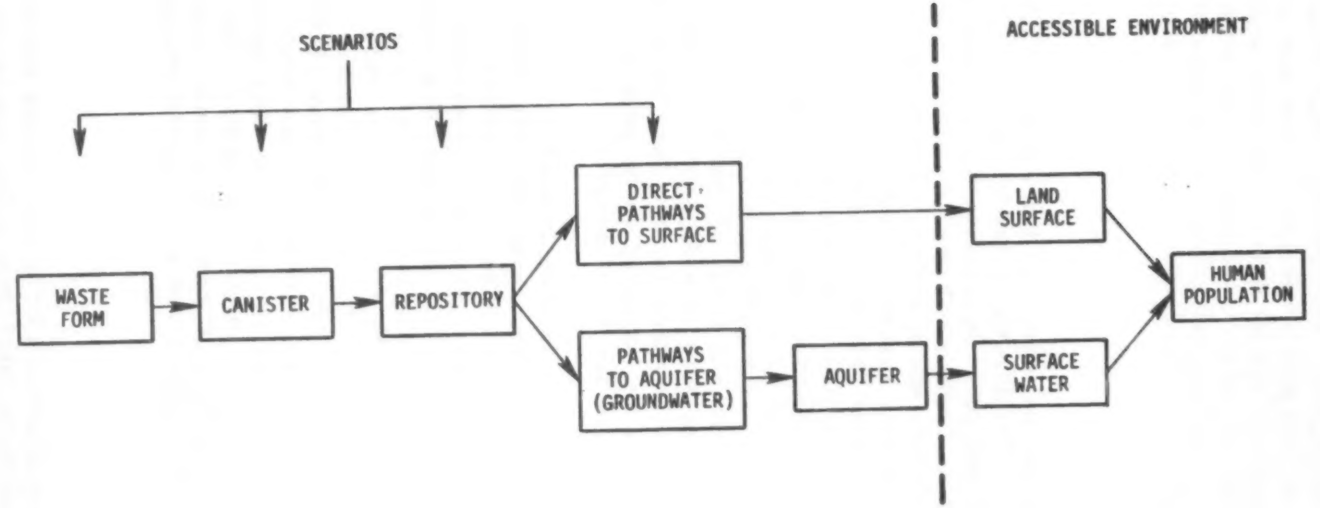


Figure 8.2-1. Components included in the risk assessment of radioactive waste releases.

The movement of radionuclides from the waste form, through the canister, through the repository, and ultimately to the accessible environment depends on a number of possible future scenarios that might alter the conditions of the underground environment. Such processes are called "release mechanisms" or "release scenarios." They may affect any of the four components indicated in Figure 8.2-1. The risk analyses reported here consider a number of potential release mechanisms. The results of the calculations for individual release mechanisms are then combined into an integrated representation of the risk from a hypothetical repository.

8.3 Time Frame

The published literature varies widely on the subject of the time frame over which radioactive waste disposal alternatives should be evaluated or compared. If the future could be predicted with reasonable assurance, then a very long time frame would provide a more complete evaluation than a short time frame, and ultimately a more complete comparison between alternative disposal systems. Some authors have attempted to compare radioactive waste disposal sites or systems for periods of up to 1 or even 10 million years. Such results, however, contain very large uncertainties because of the difficulty of making such long time-frame predictions about either the natural or the engineered components of the system. At the opposite extreme, other authors have only considered site/risk comparisons for periods on the order of 300 years, arguing that relatively reliable predictions can only be made for time frames of this length and that the longer term performance is both less important (due to the decay of the radionuclides) and expected to be generally proportional to the performance calculated for this period.

In the course of performing numerous risk assessments of radioactive waste disposal systems, the Agency has concluded that the risks identified over relatively short time spans, such as a few hundred to one thousand years, do not adequately portray important differences between alternative sites or disposal systems. This is because the ground water travel times would probably be sufficiently long at most sites that no significant radionuclide releases would be predicted over this time period. If the analyses were carried further into the future, there could be substantial differences between the sites because of the hydrologic or geochemical characteristics of their ground water systems. The primary risk assessments carried out in support of this rulemaking have thus been based on a time frame of 10 thousand years. This time frame appears long enough to identify important differences between one site and another, or between other aspects of one disposal system and another.

8.4 Measures of Risk

In considering the long-term effects of a radioactive waste repository, the Agency has considered both total cumulative population and individual effects. These two risk measures provide can different perspectives. For example, a relatively unproductive ground water supply could be contaminated with radionuclides released from a repository at some point in the future, but because of the limited availability of water from this supply, only a few individuals would be exposed to the radioactivity as a result. In this case the individual doses might be high, although the overall effect on the total population could still be quite low. On the other hand, a future release from a radioactive waste repository could lead to very low-level contamination of water supplies that serve a relatively large population. In this case the dose to any individual in that population might be small, although the overall effect could be substantial in terms of cancers or genetic effects caused in the total population. Because of these differences, the Agency has developed and applied techniques that can estimate both population and individual risks.

The mode of presentation of the risk assessment results can also vary because each release mechanism has an estimated probability of occurrence (often quite small) and the time at which the release occurs has an important influence on the population effects (due to the gradual decay of the radioactivity prior to release). One method of displaying population risks has been to assemble "complementary cumulative distribution functions" (CCDF) of resulting human health effects. A hypothetical CCDF is illustrated in Figure 8.4-1.

The CCDF shown in Figure 8.4-1 is assembled by first calculating a set of probabilities for the various release scenarios and associating the consequence (number of health effects) with each probability. Processes that normally occur (such as expected ground water flow) are treated as a scenario with a probability of one. Disruptive events are assumed to occur at several different points in time, with a corresponding estimate of probability of occurrence at that time. Once the paired probability and consequence estimates are made, they are combined into the CCDF by ranking them in the order of decreasing consequences. The relationship between probability and consequence that is generated represents a cumulative probability distribution of population risks and is displayed in Figure 8.4-1. Point A on this curve represents the large consequence of a low probability event. Point B combines the low probability event of point A with another low probability event. The probability of either of the two events occurring is greater than the probability of each event (i.e., the probability is cumulative), and the consequence is less than that given for point A. Another event probability is added to plot point C, with its associated, and lesser consequence, and so on until it is impossible to differentiate the absolute effect of an individual event, as illustrated in the region of the curve labeled D. (Where events are independent and have low probabilities, the combination of probabilities can be approximated by simple addition). The resulting curve can be interpreted at any point by the statement: "The probability shown is the probability of obtaining a consequence at least as large as the consequence shown."

Another risk measure for populations is obtained from the same set of paired probability and consequence estimates used for the CCDF. In this case, however, each pair of estimates is multiplied together and

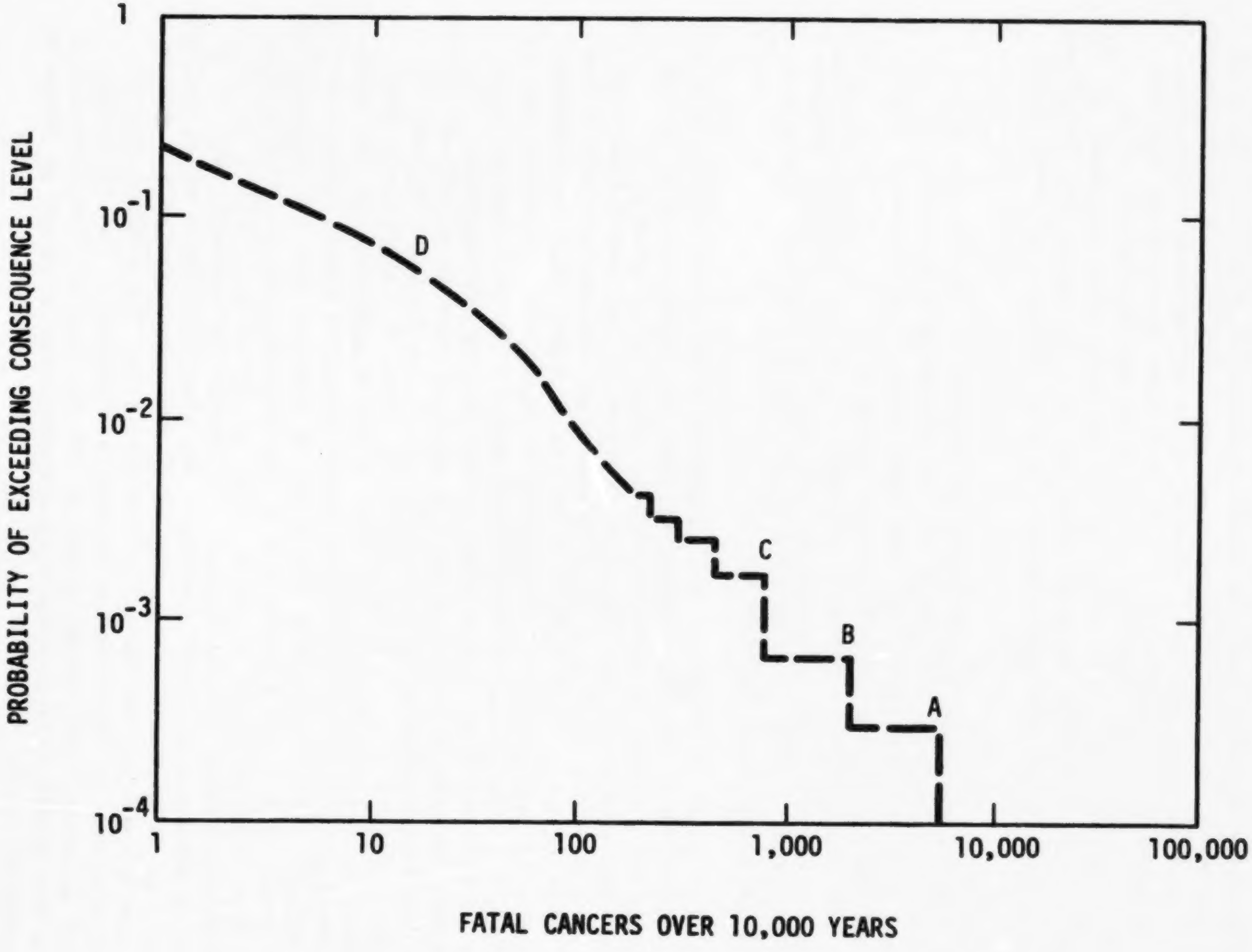


Figure 8.4-1. Hypothetical complementary cumulative distribution function (CCDF) of human health effects.

these products are summed for all the pairs. The summation results in a total of the "expected" health effects from all release scenarios from a particular repository model. This expected number of health effects corresponds to the integral under the CCDF, and it is the risk measure frequently used in this report to express the health effects associated with various types of repository sites and designs.

Finally, an individual's risk is determined by estimating the annual radiation exposure he would receive if 100 percent of his drinking water consumption (2 liters/day) was supplied from ground water contaminated by radionuclides released from a repository. This exposure is usually depicted as a function of time after disposal for an individual using water at a particular distance (typically two kilometers) from the edge of the repository along the centerline of the plume of projected contamination.

8.5 Computer Codes Utilized

A number of computer codes have been used in the Agency's risk assessments. A description of two of these codes is provided in Appendix A. The primary tool has been the program REPRISK, which has been under development at EPA since 1978 and is described in detail in Sm82. REPRISK calculates the consequences (in terms of somatic and genetic health effects and amount of radioactivity released) and probability of each of the release scenarios considered, and it combines these estimates into the total expected number of health effects caused over a period of time. REPRISK does not calulate individual exposures caused by any of the release scenarios, nor can it adequately estimate integrated releases of radioactivity for more than about 10,000 years after disposal. For calculations involving individual doses and radionuclide releases integrated over time frames longer than 10,000 years, the computer code NWFT/DVM, developed by Sandia National Laboratories under contract to the NRC, has been used. This code models the transport of decay chains whose elements have different retardation factors in the ground water system. A more complex ground water code, SWIFT, has also been used to support the EPA risk analyses, primarily to validate some of the hydrologic calculations carried out using simpler models.

8.6 Site Parameters

Models were assembled for four of the five sites tentatively identified by the DOE, in accordance with the NWPA, for nomination as potential sites for the first repository (see Chapter 4). These four sites are: 1) the bedded salt deposits in the Palo Duro basin, 2) the bedded salt deposits in the Paradox formation in Utah, 3) the basalt flows on the Hanford reservation in Washington, and 4) the unsaturated volcanic tuff formations at Yucca Mountain in Nevada. In addition, the Agency assembled several models of repositories in granitic formations. Two of these attempt to roughly represent geological and hydrological conditions that might be encountered in certain regions of the country: 1) a model for the north-central portion of the United States (granite I), and 2) a model for the New England area (granite II).

In both of these cases, descriptive parameters were selected that are likely to overestimate the risks from disposal in these areas. More research and careful site selection should be able to identify sites that would perform better than these models. For comparison, the Agency also used the idealized model of a granite repository that was considered for the proposed rule (granite III). This model is typical of the objectives that might be set in looking for a very protective site in granite and may represent conditions that would be difficult to find; however, the Agency believes that this third model provides a useful perspective because it indicates the range of performance attributable to repositories in granitic media.

A number of parameters are used to describe the geometry and hydrologic conditions assumed for each of these seven model repository sites. These are listed in Table 8.6-1 and can best be understood in conjunction with the generic cross section shown in Figure 8.6-1, with certain exceptions to be described shortly. The conceptual framework of the lithology at the site is that the repository is located between two aquifers called, respectively, the "upper aquifer" and the "lower aquifer." To simulate conditions present at real sites, the upper and lower aquifer do not generally represent single hydrostratographic units, but rather represent "synthetic aquifers" whose properties are defined to approximate the combined properties of a number of transmissive units above and below the repository horizon. For example, if a number of such transmissive units are present above the repository at a particular site and if the application of the generic models described here is intended to represent conditions similar to those at that site, then one can calculate the combined volumetric flows in the upper units and define appropriate hydrologic parameters so that the synthetic aquifer represents the same total flow. Similarly, by varying one or more additional parameters, it is possible to simulate the effective fluid velocity in any one of the actual units. This will be illustrated in subsequent sections when specific lithologies are discussed.

The general way in which the REPRISK model constrains the risk analyses is that the upper aquifer is considered to be the pathway of ground water release of any radionuclides that leave the repository. (An upward gradient is considered to exist along any pathway that might be present or develop between the lower aquifer and upper aquifer.) This is why greater emphasis is placed on the properties of the upper aquifer in Table 8.6-1. At potential repository sites, however, the hydrogeologic environment may be different from that assumed in the generic model. For example, there may be no significant aquifer below the repository (as in a number of crystalline rock sites), or above the repository (as in the case of a repository in the unsaturated zone), or there may be a prevailing gradient that is downward from the upper aquifer, in which case the lower aquifer would appear to be the more likely release pathway. These cases can all be accommodated within the modeling framework of REPRISK.

With respect to the specific hydrologic parameters listed in Table 8.6-1, hydraulic conductivity is used in conjunction with Darcy's Law to

Table 8.6-1. Site parameters considered in risk assessment

Parameter	Basalt	Palo Duro Bedded Salt	Paradox Bedded Salt	Tuff	Granite I (N.Central)	Granite II (New England)	Granite III (idealized)
Distance from repository to overlying aquifer, meters	22	1105(a)	666	100(a)	448	370	230
Hydraulic conductivity of the host rock between the repository and the aquifer, after thermal effects (centimeters/sec)	10^{-7}	0	0	$3x10^{-9}$	10^{-7}	10^{-7}	10^{-9}
Porosity of rock between the repository and aquifer	10^{-4}	n/a	n/a	0.1	10^{-4}	10^{-4}	10^{-4}
Natural hydraulic gradient between the repository and aquifer	0.025	0.26^{a}	0	1.0^{a}	0	0	0
Thickness of aquifer, meters	30	300	20	1000	10	80	30
Hydraulic conductivity of aquifer (centimeters/sec)	10^{-3}	$5x10^{-6}$	$2x10^{-5}$	10^{-4}	$2x10^{-5}$	10^{-3}	10^{-4}
Porosity of aquifer	0.01	0.05	0.2	0.002	0.018	0.039	0.15
Horizontal gradient in aquifer	0.0003	0.005	0.02	0.0003	0.055	0.01	0.01
Horizontal distance along the aquifer to the accessible environment, meters	2000	2000	2000	2000	0^{b}	2000	2000

(a) For these sites, ground water flow is assumed to be downward to the lower aquifer, and all aquifer characteristics are those of the underlying aquifer.

(b) The model for this site assumes that surface water on the site is in contact with the ground water; therefore, there is no horizontal distance to the accessible environment, but only a vertical flow path.

Source: EPA85

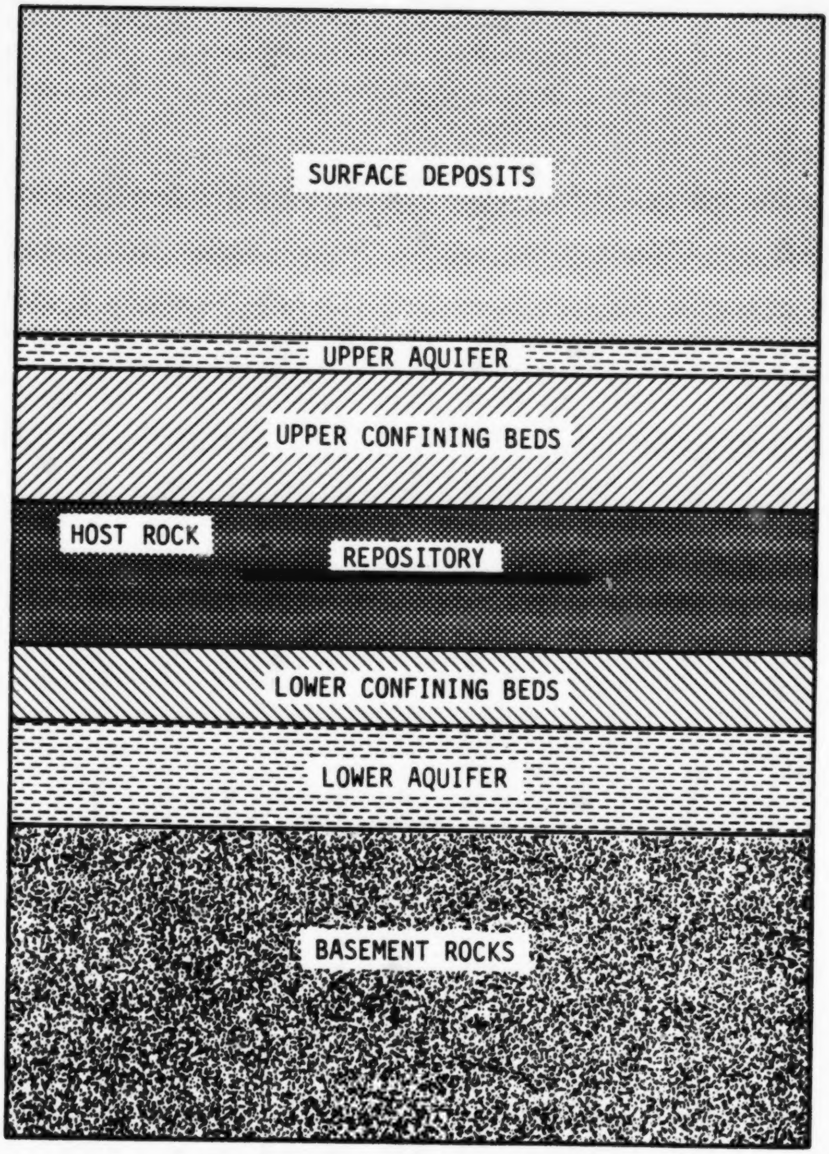


Figure 8.6-1. Cross section of the rock formation at the generic repository site.

estimate volumetric flow rates through various components such as pathways from the repository to the upper aquifer or along the upper aquifer itself. For further elaboration on the mathematical equations referred to, one may consult EPA80 and the references cited there. Only Darcian flow has been treated in the analyses, and work by DOE at specific sites tends to confirm that the flow regimes are such that this approach is adequate. The porosity is used to convert volumetric flow rates into average effective fluid velocities in the direction of movement. In particular, the volumetric flow rate is divided by porosity to obtain an effective fluid velocity. It is important to make this distinction when considering the time of arrival of contaminated ground water at the discharge point to the accessible environment. Hydraulic conductivities specified as part of the description of a conceptual site may be modified in the course of the modeling as a result of other phenomena assumed to be present, as described later in the section on release mechanisms. The vertical gradient between the upper and lower aquifers may be modified because the thermal effects of the buried waste change the density of ground water and create buoyancy effects. The same thermal effects modify the viscosity of groundwater in the vicinity of the repository, and thereby enhance the effective hydraulic conductivity.

Geochemical characteristics constitute another important set of site parameters. These include the ways in which the movement of any radioactivity released from the waste would be slowed or "retarded" by geochemical interactions with the surrounding rocks, and the expected limits that the geochemical conditions would place on the solubility of many of the radioactive elements in the waste. Table 8.6-2 lists the retardation factors and solubility limits used in each of the repository models for the elements of primary interest. The retardation factors describe the relative speed that the respective radionuclides travel in ground water compared with the speed of the ground water itself (i.e., an isotope with a retardation factor of 10 moves one tenth (1/10) as fast as the ground water). The methods by which REPRISK utilizes retardation values has been described previously (Sm82); they assume that dispersion can be neglected in the calculations. This same assumption has been used extensively in the literature.

8.7 Repository Parameters

Certain assumptions need to be made pertaining to the geometry and physical characteristics of the repository itself. The DOE has frequently modified its conceptual designs for repositories, and the parameters assumed by the Agency, based on DOE publications from several years ago, may not reflect the most current engineering plans. Nevertheless, an examination of the Agency's risk analysis models indicates that they are not highly sensitive to these engineering assumptions. Therefore, the Agency has adopted a single set of assumptions to be applied to repositories at all potential types of sites. These parameters, as well as their actual numerical values, are shown in Table 8.7-1. The repository considered is assumed to contain 100,000 metric tons of heavy metal (MTHM) as spent nuclear fuel.

Table 8.6-2. Geochemical parameters used in risk assessment

Parameter	Basalt	Palo Duro Bedded Salt	Paradox Bedded Salt	Tuff	Granite II
Retardation Factors[a]					
carbon	1	1	1	1	1
strontium	200	10	10	200	200
zirconium	5000	1000	1000	5000	5000
technetium	5	5	5	5	5
tin	1000	100	100	1000	1000
iodine	1	1	1	1	1
cesium	1000	10	10	500	1000
uranium	50	20	20	40	50
neptunium	100	50	50	100	100
plutonium	500	200	200	200	200
americium	500	1000	1000	1000	3000
Solubility Limits[a] (parts per million)					
carbon	none	none	none	none	none
strontium	0.6	none	none	none	none
zirconium	0.0001	0.0001	0.0001	0.0001	0.000001
technetium	0.001	0.001	none	none	none
tin	0.0001	0.001	0.001	0.0001	0.0001
iodine	none	none	none	none	none
cesium	none	none	none	none	none
uranium	0.001	0.01	0.01	none	0.001
neptunium	0.0001	0.001	0.001	0.1	0.1
plutonium	0.00001	0.001	0.001	0.001	0.001
americium	0.001	0.1	0.01	0.001	0.001

[a] For use with all isotopes of radionuclides listed.

Source: EPA85

Table 8.7-1. Repository parameters considered in risk assessment

Parameter	Value
Dimensions of repository:	
Length:	4,000 meters
Width:	2,000 meters
Height:	5 meters
Total mined-out volume:	10^7 meters^3
Average porosity of backfilled repository.	0.2
Time to maximum backfill compaction due to plastic flow (salt only)	200 years
Number of canisters of high-level waste:	35,000
Number of waste drifts:	350
Canisters per drift:	100
Length of waste drift:	500 meters
Canister spacing:	5 meters

Source: EPA85

Because of the rectangular dimensions of the repository, an assumption must be made about the orientation of the repository with respect to flow direction in the upper aquifer. The longest dimension of the repository, 4 km, is assumed to be in the same direction as this flow pattern. The mined volume of the repository, as well as the porosity of the backfill, must be considered in calculating the amount of radionuclides that might dissolve in the ground water that would gradually seep into the repository after its closure. Because such dissolution might be limited by solubility factors, this water volume is significant because it can be used to estimate the amount of dissolved radionuclides that might be withdrawn by an exploratory well that penetrates the repository at some point in the future.

The case of a salt repository is different because it would be expected to gradually seal after closure as a result of salt creep. In this case, therefore, the time to closure and the amount of moisture present at that time are important for the risk analysis. The other parameters in Table 8.7-1 are used in connection with specific release mechanisms and will be discussed in subsequent sections.

8.8 Waste Package Parameters

For these assessments, the waste package is assumed to consist of two main components: the waste form and the waste canister. Each of these may actually be quite complex. For example, the waste form consists of an initial inventory of radionuclides contained in a physical matrix, which may be unreprocessed spent fuel, borosilicate glass, or some other alternative. Thus, the waste form might have a homogeneous physical structure, as in the case of glass, or a more complicated one. The canister may actually consist of a number of components, such as a mostly concentric set of containers with spaces, coatings, or other materials between them. For the conceptual analyses reported here, the Agency has decided to adopt a simple model for the waste package, consisting of a homogeneous waste form and a single canister.

Table 8.8-1 shows the assumed initial inventory of radionuclides, which is based on detailed calculations and considerations described in the risk assessment for the proposed rule (Sm82). The radionuclide inventory of a waste will vary according to whether the waste consists of spent fuel itself or whether the spent fuel has been reprocessed to recover some of the transuranic components. Since reprocessing is not being pursued for commercial nuclear power applications, a radionuclide inventory that is equivalent to that associated with direct disposal of spent fuel has been assumed. The choice of radionuclides listed in Table 8.8-1 is not all inclusive, but it includes the dominant radionuclides as well as a range of radionuclides with different properties. Thus, a risk assessment based on the radionuclides shown provides an adequate representation of the risks associated with a repository.

The canister is a protective container assumed to inhibit the leaching or the disolution of the waste form and the consequent transport of

Table 8.8-1. Radionuclide inventory in repository (spent nuclear fuel)

Radionuclide	Initial Quantity in Repository (ADL79) (curies)	Half-Life (years)
C-14	28,000	5,730
Sr-90	6,000,000,000	29
Zr-93	190,000	950,000
Tc-99	1,400,000	210,000
Sn-126	56,000	100,000
I-129	3,800	16,000,000
Cs-135	22,000	3,000,000
Cs-137	8,600,000,000	30
U-234	190,000	250,000
Np-237	33,000	2,140,000
Pu-238	220,000,000	89
Pu-239	33,000,000	24,400
Pu-240	49,000,000	6,260
Pu-242	170,000	380,000
Am-241	17,000,000	458
Am-243	1,700,000	7,650

radionuclides toward the accessible environment. In these risk assessments, the performance of the canister has been conservatively approximated by a single lifetime. Up until this time is reached, no radionuclides are assumed to be released from undisturbed waste packages. Once the lifetime is reached, the cansiter is neglected in the subsequent analysis, thus ignoring any benefits the remaining portions of the canister might offer.

The release rate of radionuclides from the waste form, after the beginning of the period when the canister can no longer be relied upon, may be governed by one of two limiting processes. The first of these processes is leaching, which is controlled by the accessibility of the radionuclides within the waste form to any ground water with which the waste form may come into contact. A leach rate, specified in terms of fraction leached per year, is the input parameter used to characterize this aspect of radionuclide release. The second limiting process is that of solubility. Even though the ground water may have access to radionuclides within the waste form matrix, the water may already contain sufficient amounts of the dissolved material so that no more can enter into solution without corresponding precipitation, resulting in a constant concentration. Such solubility limits for individual radionuclides are also taken into account in the risk calculations reported here, as discussed in Section 8.6.

8.9 Release Mechanisms

The release mechanisms through which radioactive wastes may escape the repository were introduced in Section 8.2. These release mechanisms may lead to the direct transport of radionuclides to the land surface or to the atmosphere, or they may lead to the ground water transport of waste away from the repository.

The purpose of this section is to review each of the release mechanisms and summarize the conceptual models that have been used in the risk assessment. The equations used to implement the conceptual models are discussed in the risk assessment that supported the proposed rule (Sm82). Further background on the release mechanisms can be found one of the other technical support documents for the proposed rule (ADL79). The six classes of release mechanisms considered by REPRISK for these risk assessments, together with the parameters describing them, are shown in Table 8.9-1.

8.9.1 Normal Ground Water Flow

Except for the case of a repository in salt, normal ground water flow refers to the movement of water through the repository horizon according to the natural hydrologic conditions, perturbed to some degree by the presence of the repository. During the construction and operation of the repository, water in the surrounding rock would be expected to gradually drain so that the rock will enter an unsaturated conditon near the openings. (In the case of tuff, the repository would be located in a rock mass that would be unsaturated at the start.) After the end of the

Table 8.9.1. Release mechanism parameters considered in risk assessments

Parameter	Basalt	Bedded Salt	Tuff	Granite
Normal Ground Water Flow				
Fraction of the repository with which ground water can communicate	1.0	-	1.0	1.0
Hydraulic conductivity of flow path (centimeters/sec)	10^{-7}	0.0	$3\text{x}10^{-9}$	10^{-7} (I,II) 10^{-9} (III)
Porosity of flow path	0.0001	-	0.1	0.0001
Cross-sectional area of flow path (square meters)	$8\text{x}10^{6}$	-	$8\text{x}10^{6}$	$8\text{x}10^{6}$
Probability of occurrence	1.0	-	1.0	1.0
Fault Movement				
Fraction of the repository with which ground water can communicate	1.0	$5\text{x}10^{-2}$	1.0	1.0
Hydraulic conductivity of flow path (centimeters/sec)	10^{-2}	10^{-4}	$6\text{x}10^{-8}$	10^{-2}
Porosity of flow path	0.1	0.1	0.1	0.1
Cross-sectional Area of flow path (square meters)	4000	4000	4000	4000
Frequency of occurrence (per year)	$3\text{x}10^{-5}$	10^{-6}	$8\text{x}10^{-5}$	10^{-6}

Table 8.9-1. Release mechanism parameters considered in risk assessments (continued)

Parameter	Basalt	Bedded Salt	Tuff	Granite
Breccia Pipe Formation				
Fraction of the repository with which ground water can communicate	-	$1.6x10^{-2}$	-	-
Hydraulic conductivity of flow path (centimeters/sec)	-	10^{-2}	-	-
Porosity of flow path	-	0.2	-	-
Cross-sectional Area of flow path (square meters)	-	30000	-	-
Frequency of occurrence (per year, after 1000 years)	0	10^{-8}	0	0
Drilling (does not hit a canister)				
Fraction of the repository with which ground water can communicate	1.0	$6x10^{-5}$	0	1.0
Volume of ground water brought to the surface	200	1.14	-	200
Frequency of occurrence (per year after 100-year control period ends)	10^{-3}	$2x10^{-2}$	-	$2.5x10^{-3}$

Table 8.9-1 Release mechanism parameters considered in risk assessments (continued)

Parameter	Basalt	Bedded Salt	Tuff	Granite
Drilling (hits a canister)				
Fraction of one cansiter brought to the surface	0.15	0.15	0.15	0.15
Frequency of occurrence (per year after 100-year control period ends)	10^{-5}	2×10^{-5}	2.5×10^{-6}	2.5×10^{-6}
Volcanoes				
Fraction of the repository inventory dispersed	4×10^{-4}	4×10^{-4}	4×10^{-4}	4×10^{-4}
Frequency of occurrence (per year)	6×10^{-10}	10^{-10}	3×10^{-8}	10^{-10}

Source: EPA85

operational period and sealing of the repository, water would be expected to gradually seep back into pores and fractures in the rock and establish a flow regime connected to the regional ground water system.

The resulting flow patterns may be different from those prior to the excavation of the repository. For example, the heat generated by the waste may modify the hydraulic conductivity of the surrounding rock and may also change the properties of water, making it less dense and less viscous. The lower density can lead to a buoyancy effect that may cause an increased vertical hydraulic gradient. The decreased viscosity enables the water to flow more easily through the rock and hence allows for potential increases in flow rates.

In the case of a repository in tuff, normal ground water flow refers to the downward percolation of water through the unsaturated rock toward the water table. This downward movement is not expected to be influenced greatly by the presence of the repository because the limiting factor is essentially the amount of water itself.

In the case of a salt repository, the salt formations themselves are assumed to be effectively impermeable to ground water movement. Therefore, "normal" ground water flow has been used to characterize a number of processes, such as leakage along repository shafts, which are grouped under this category because they are treated in the same part of the REPRISK model. These mechanisms were reviewed in the risk assessment supporting the proposed rule and were generally not expected to cause releases within 10,000 years after disposal (Sm82).

8.9.2 Fault Movement

The faulting release mechanism covers both the cases of a new fault occurring at a repository site (and intersecting the repository itself) as well as the case of the reactivation of an old apparently stable fault. This reactivation is assumed to lead to an intersection with the repository itself, which is treated in the model as a vertical planer structure with increased hydraulic conductivity over and above that of the original rock. Thus, a fault can lead to a new and preferential pathway for ground water movement. A fault's hydrological properties are different from those of the original rock and, in fact, rather high volumetric flows could pass through such features. In addition to creating a flow pathway, the model for faulting assumes that this can be a relatively violent and disruptive event, destroying the integrity of waste packages within a certain distance of the fault. The result can be an earlier onset of leaching (if this had not already begun by the time of fault movement). As in the case of normal ground water flow, the only releases by faulting are assumed to be via a pathway connecting the repository with an aquifer, thereby enabling ground water transport of the waste. Faulting has been treated as a random stochastic process for purposes of the Agency's risk assessments. This is not to say that faulting is a random process, but only that faulting at a real respository site should be able to be bounded by modeling it as a physical process that occurs randomly. The likelihood of new or reactivated faults is estimated on the basis of geometric arguments and simple probability concepts (ADL79).

8.9.3 Breccia Pipe Formation

The breccia pipe release mechanism refers to the development of a localized dissolution feature and only applies to the case of a repository in salt. Breccia pipes, or collapse chimneys, have been known to develop from the base of the salt beds and proceed vertically in a relatively rapid and sometimes abrupt fashion. Such a breccia pipe is assumed to provide a relatively high permeability zone that can facilitate the vertical movement of ground water. In addition, it is assumed to violate the integrity of waste packages that are located within the collapse zone. A probability of occurrence of such features is based on observed statistical averages and is assumed to occur randomly. Since good site-selection procedures should enable the selection of a site where the likelihood of occurrence of a breccia pipe is much less than the average occurrence rate, the resulting calculation is believed to be conservative (i.e., tend to overestimate the risk).

8.9.4 Inadvertent Intrusion by Exploratory Drilling

Future exploratory drilling at a repository site cannot be ruled out, even though steps will be taken to signal to future generations that dangerous materials are buried there. The Agency has considered a wide range of potential purposes for drilling in different geologic media and has estimated drilling rates that are intended to be upper bounds on the future likelihood of drilling at a repository site. In estimating these values, no credit has been taken for the communication to future generations of the presence of the repository, except that for 100 years after disposal it is assumed that such communication would be completely effective.

The main purposes for future drilling may vary from one kind of geology to another. For example, for salt deposits in sedimentary basins, the dominant drilling is expected to be in search of oil and gas, whereas in a granitic terrain the dominant purpose might be exploration for water or minerals (ADL79). The drilling release mechanism contains a number of components. First, it is possible that the drill hole would actually intersect a waste canister, with the result that waste materials could be brought directly to the surface with the drilling mud. In this case, such materials would likely go unnoticed or unrecognized, at least for some time, and hence they could be distributed on the land surface. Even if a drill hole does not intersect a waste package itself, if it occurs sufficiently far in the future so that the waste packages have been attacked by ground water and waste materials have begun to be leached out, the drilling fluid could carry some of these dissolved radionuclides to the surface. This is especially the case if the drillers recognize that they had passed through a relatively porous zone (the backfilled repository) and pumped water from that level in order to see if it might be a suitable source of water. This mechanism would also lead to the release of radionuclides directly to the land surface. A third method by which future drilling could lead to the release of radionuclides from a repository is that the decommissioned borehole, after being filled and perhaps sealed, might still represent a relatively permeable pathway from the repository to adjacent water-bearing zones. This could facilitate the flow of ground water through the repository and the release of radionuclides to an aquifer. In the calculations reported in this document, this third release mechanism has not been included because earlier calculations showed that its contributions were negligible by comparison to other forms of release (Sm82).

8.9.5 Volcanism

Volcanism also has the potential to release waste from a repository, either by transporting it directly to the surface or by translocating it in an underground volcanic structure, such as a sill or a dike, which may also encounter an aquifer. Earlier calculations indicated that this latter mode of transport is overwhelmingly dominated by the fault release mechanism, in terms of likelihood of occurrence, and that it is roughly similar in terms of consequences (ADL79). Therefore, it has not been included in these assessments because its contribution is negligible. The release of radionuclides to the surface, however, by way of magma, ash, or gases passing vertically upward through the repository has the potential for significant distribution of radioactivity to the accessible environment, even though its likelihood is small. Therefore, it has been included in the calculations for certain lithologies that have been investigated in relatively close proximity to active volcanic regions. This does not mean that the Agency believes that volcanism is likely at any of the sites that are being investigated, but only that it may be the dominant low probability, high consequence event and hence should be included in the calculations to give an adequate perspective on the spectrum of risks. In the case of the release of radionuclides by volcanism, the model has components to deal with waste releases to both the air and land surface.

8.10 Risk Assessments for Models of Geologic Repositories

Using the approach summarized in the previous sections, the Agency estimated the long term risks to future populations and individuals from disposal of high-level radioactive wastes in several different types of mined geologic repositories.

8.10.1 Population Risks

One set of analyses used the REPRISK program to estimate the long-term population risks over 10,000 years from four models that the Agency believes are representative of most of the sites being considered by DOE as candidates for the first repository. A related set of analyses considered the long term population risks from three conceptual models of the granite formations that DOE is beginning to assess as part of the program to identify sites for the second repository called for by the NWPA. The various models of granite formations do not represent particular sites because sufficient information is not yet available for such analyses.

Instead, they represent a range of possibilities based on regional data and generalized expectations about granitic formations. For these "reference case" assessments, all of the models assume a waste package lifetime of 300 years and a subsequent waste form release rate of one part in 100,000 (10^{-5}) per year, which are the minimum requirements of NRC's 10 CFR Part 60.

Figures 8.10-1 and 8.10-2 summarize the results of these various assessments of long-term population risks. Both figures display the same information; however, Figure 8.10-1 shows the population risks on a linear scale, while Figure 8.10-2 introduces the logarithmic scale that will be used for many of the results in this chapter. Figure 8.10-2 also highlights the risk level of 1000 fatal cancers over 10,000 years that has been used as a basis for the containment requirements (Section 191.13) in the final 40 CFR Part 191. Finally, Figure 8.10-2 indicates (where applicable) the corresponding risk estimates developed in the technical basis for the proposed rule; this perspective demonstrates how the analytical revisions made in response to the SAB review have resulted in substantially lower population risk estimates for the various geologic media considered. As Figures 8.10-1 and 8.10-2 show, there is a considerable variation between the risks projected for different types of geologic media; however, all of these reference case estimates are well below the level of protection sought by the containment requirements.

Table 8.10-1 provides more detail on these assessments by displaying the risks from the four most important release mechanisms for several of the geologic media, with the dominant radionuclides for each release mechanism also displayed.

For the crystalline rocks below the water table (i.e., basalt and granite), where some amount of normal ground water flow is expected, this release mechanism dominates the long-term risks. The radionuclides that flow most readily with the ground water, such as carbon-14 and iodine-129, cause most of this long-term impact for the basalt model. For the granite II model, which has very rapid ground water flow, neptunium-237 and the plutonium isotopes contribute significantly, despite their moderate retardation in the ground water. In media where such normal ground water flow is not expected, different release mechanisms dominate. For the bedded salt models, risks from inadvertent human intrusions that bring contaminated water to the surface are the most important contributors to the long-term risks. For the tuff model, fault movement is the most important contributor because fault movement can initiate increased ground water flow, while inadvertent intrusion is much less likely to bring contamination to the surface than it is for the other models because the repository horizon would not be saturated with ground water.

Figures 8.10-3 and 8.10-4 display the long-term population risks in another way. For the four models of potential first repository sites, these figues show the complementary cumulative distribution functions of the population risks over 10,000 years, as discussed in Section 8.5. The figures also indicate corresponding consequences in terms of the ratios of the projected radionuclide releases to the release limits specified in

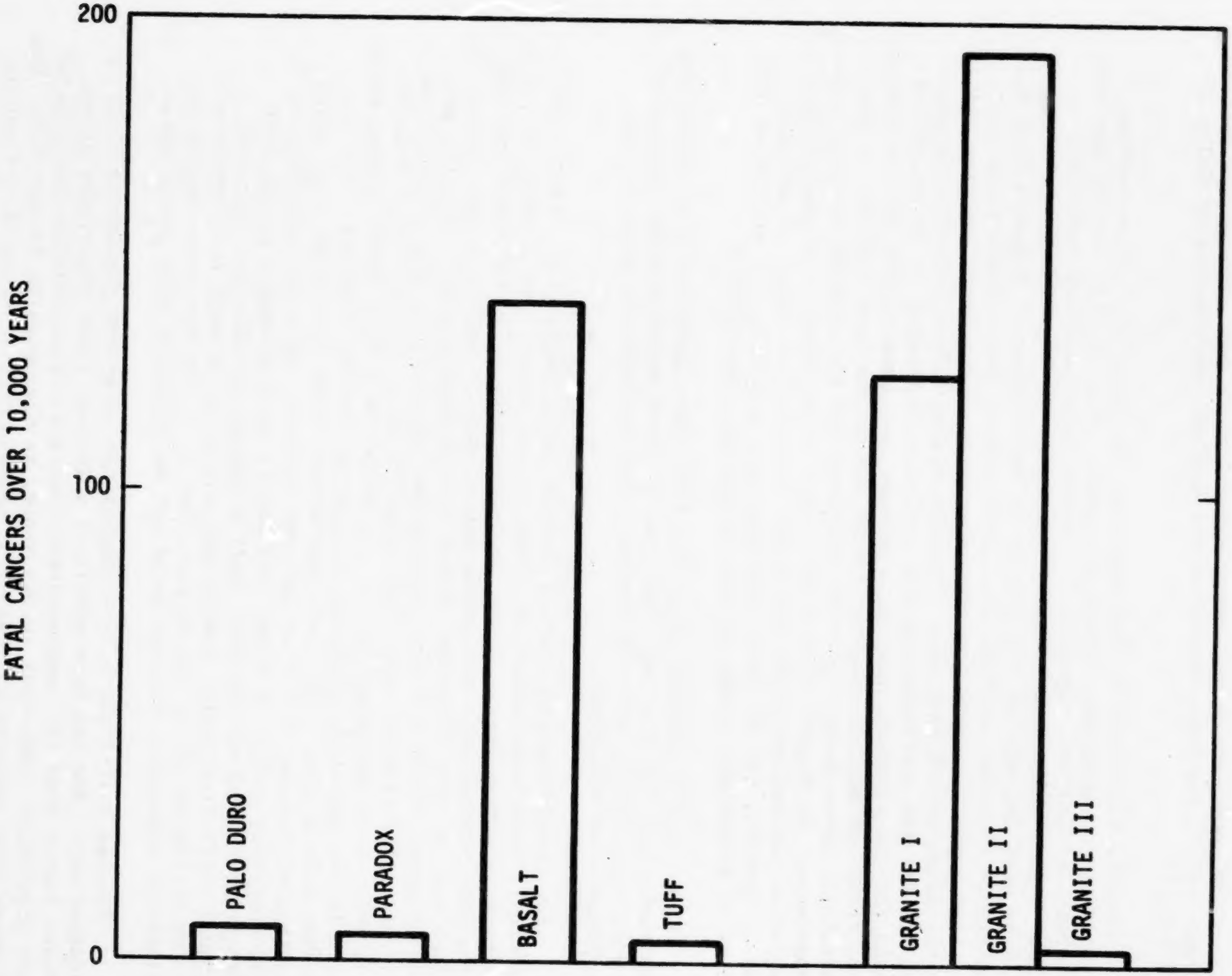


Figure 8.10-1. Population risks from disposal in geologic repositories (reference cases).

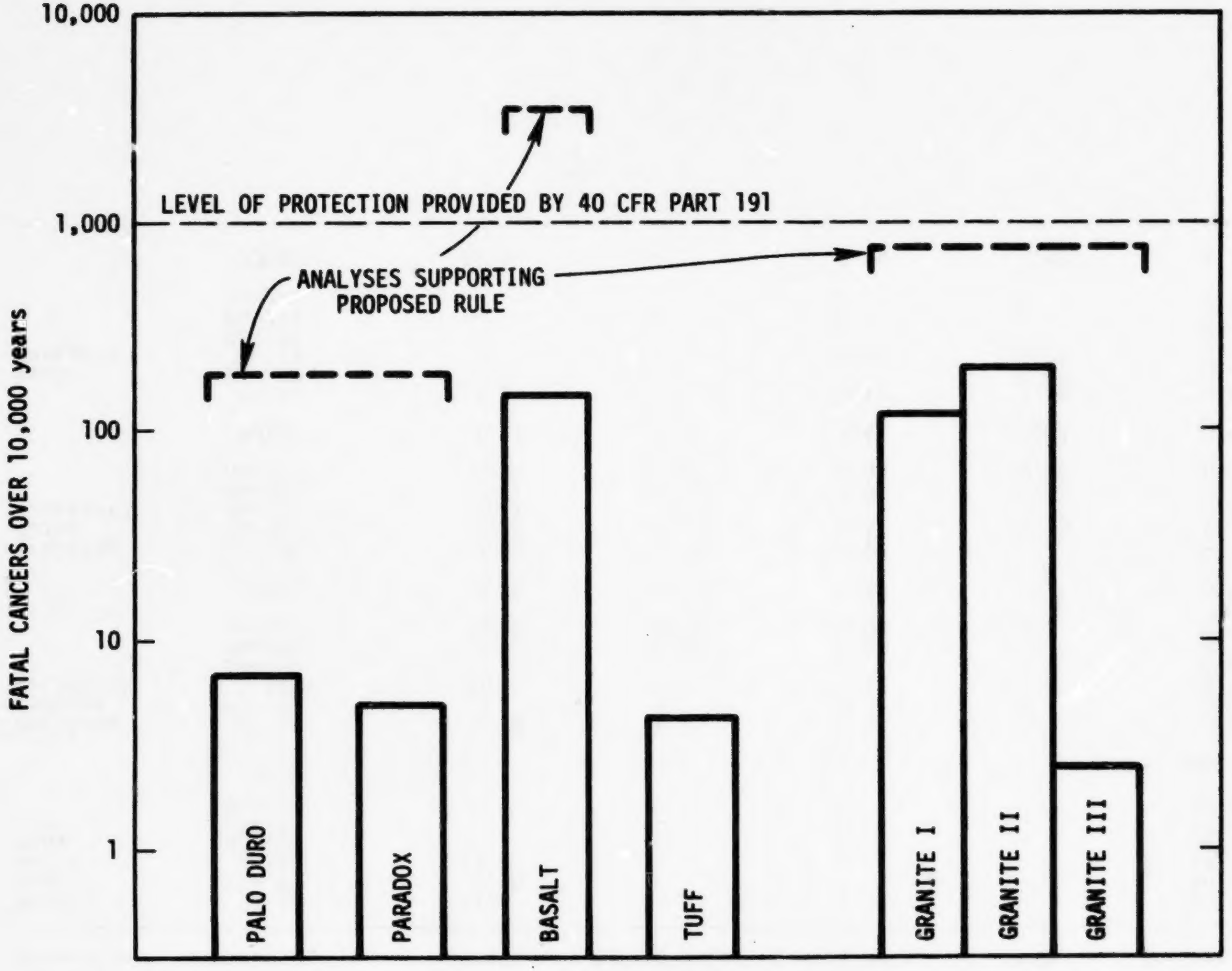
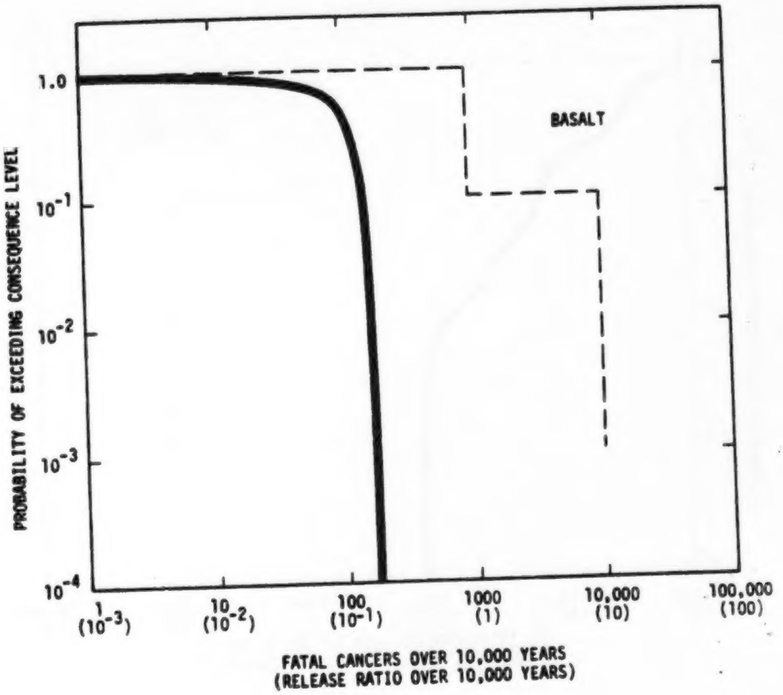


Figure 8.10-2. Population risks from disposal in geologic repositores (logarithmic scale, reference cases).

Table 8.10-1. Fatal cancers over 10,000 years by release mechanism and radionuclide

Release Mechanism	Nuclide	Basalt	Palo Duro Bedded Salt	Paradox Bedded Salt	Tuff	Granite II
Normal Ground Water Flow	C- 14	71.1	-	-	-	52.5
	Tc- 99	0.56	-	-	-	30.6
	I-129	25.3	-	-	-	18.8
	Np-237	-	-	-	-	38.1
	Pu-239	-	-	-	-	12.5
	Pu-240	-	-	-	-	29.9
	TOTAL	97.0	-	-	-	184.0
Drilling (misses canister)	C- 14	0.29	0.41	0.41	-	0.09
	Tc- 99	-	-	0.27	-	0.06
	I-129	0.12	0.17	0.17	-	0.03
	Pu-240	-	-	-	-	0.05
	Am-241	1.61	2.20	0.29	-	0.51
	Am-243	0.22	0.27	0.03	-	0.06
	TOTAL	2.30	3.16	1.29	-	0.92
Drilling (hits canister)	Pu-238	0.02	0.03	0.03	-	-
	Pu-239	0.58	1.13	1.13	0.15	0.15
	Pu-240	0.59	1.16	1.16	0.15	0.15
	Am-241	0.51	1.01	1.01	0.13	0.13
	Am-243	0.03	0.05	0.05	0.01	0.01
	TOTAL	1.73	3.41	3.41	0.44	0.44
Fault Movement	C- 14	17.1	-	0.11	1.66	0.69
	Tc- 99	0.1	-	-	0.59	0.46
	I-129	7.2	-	0.05	0.75	0.28
	Np-237	-	-	-	-	0.32
	Pu-239	-	-	-	-	2.12
	Pu-240	-	-	-	-	4.33
	TOTAL	24.4	-	0.16	3.00	8.49



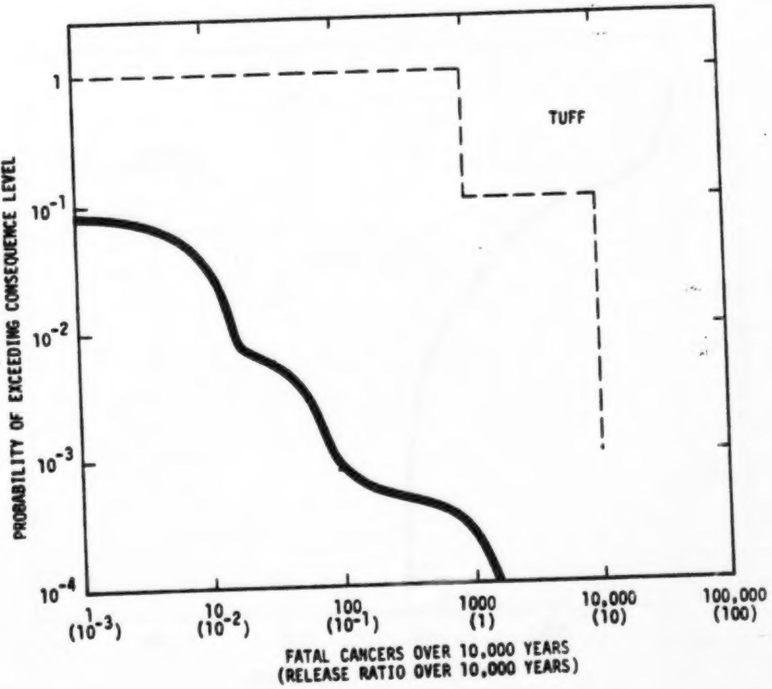
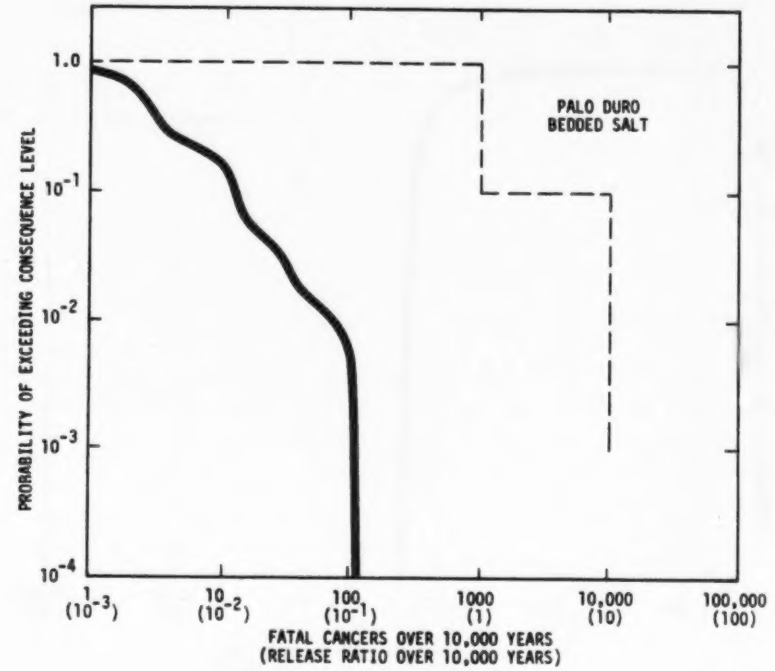


Figure 8.10-3. Complementary cumulative distribution functions of the population risks for disposal in basalt and tuff.



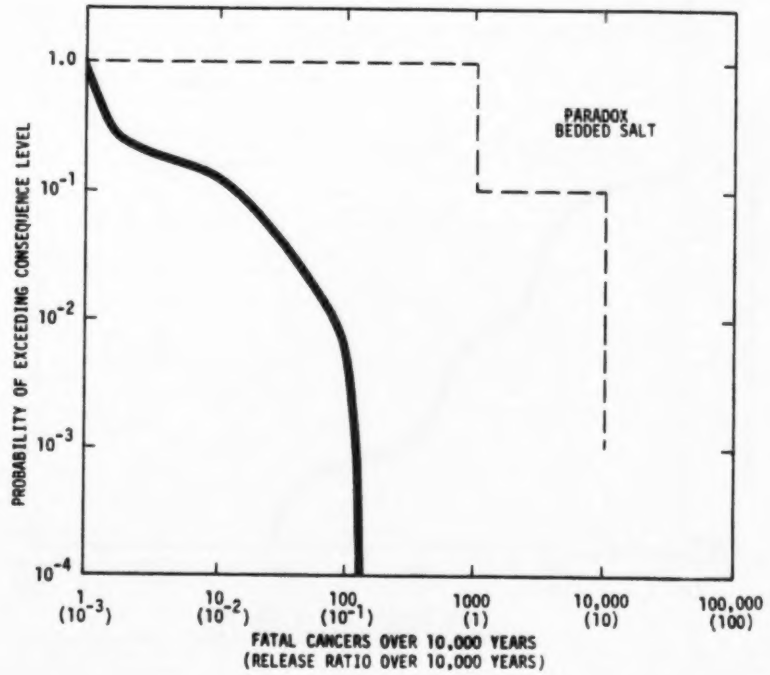


Figure 8.10-4. Complementary cumulative distribution functions of the population risks for disposal in bedded salt.

Section 191.13 of the final rule. Finally, the figures depict the probability and consequence limits that must not be exceeded in order to comply with Section 191.13.

8.10.2 Individual Risks

The NWFT/DVM program was used to estimate the individual radiation exposures to people who might use the ground water in the overlying aquifer for drinking water at a distance of 2 km from the outer edge of a repository. Only the potential exposures due to releases from expected ground water flow were evaluated; no disruptive events were considered (this is "undisturbed performance" as defined in the final rule). Of the models corresponding to sites being considered for the first repository, only the model for Hanford projects releases for expected ground water flow within 10,000 years after disposal. Therefore, only individual exposures for the Hanford model are discussed in this section. The same site, repository, and waste package parameters were used as for the population risk assessments summarized in the preceding section, including a 300-year waste package lifetime and a 10^{-5} waste form release rate.

Figure 8.10-5 displays the results of these individual exposure calculations. After 300 years, when the waste package is assumed to fail, ground water from the repository containing radionuclides from the waste would begin to travel to and along the overlying aquifer. For the hydrological parameters assumed in the basalt model, a little more than 200 years would be required for such ground water to travel along the aquifer. Thus, potential exposures would begin to appear soon after 500 years after disposal (300 years for the canister lifetime plus 200 years for ground water travel). The maximum projected exposure would quickly rise to about 2 rems/year (for an individual obtaining all of his drinking water from the center of the plume of contamination in this aquifer) due to releases of iodine-129 and carbon-14. This exposure level would remain approximately constant to almost 10,000 years, when the first appearance of other, slower-moving radionuclides would cause the maximum concentration to begin to rise.

8.11 Uncertainties in the Risk Assessment

The results of the risk assessments discussed in Section 8.10 encompass many uncertainties, which are due to a number of factors such as the following:

- The long time frame over which predictions are needed.
- The simplified nature of the models in comparison with the real physical situation.
- The generic nature of the modeling, i.e., no detailed site-specific data for a particular real site.

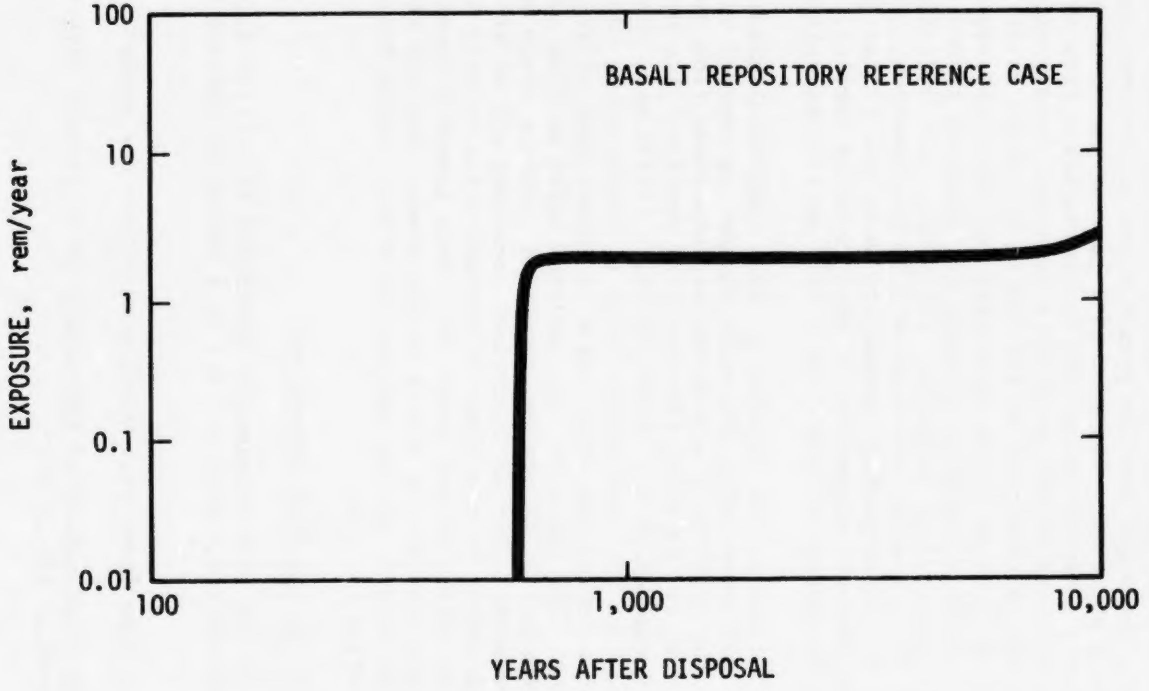


Figure 8.10-5. Radiation exposures from drinking ground water at a 2-kilometer distance from a repository.

The purpose of the risk assessment is to make rough approximations of the capabilities of geologic disposal of radioactive waste. Therefore, despite these uncertainties, the Agency believes that the estimates generated herein provide an adequate technical basis for the associated regulations.

In order to lend perspective to the uncertainties in these calculations, the Agency has proceeded as follows. First, in estimating parameters or in choosing models to represent various processes, an attempt has been made to consistently overestimate factors that contribute to risks from the repository. This is the same philosophy that was adopted in risk assessments for the proposed rule, although the degree of overestimation has been reduced in reponse to many of the recommendations of the Agency's Science Advisory Board.

Second, extensive use has been made of sensitivity analyses in order to understand how much the results of the assessments vary with the variation in certain model components or parameters. For parameters that are particularly important in determining the final risk results, special attention has been devoted to choosing appropriate values.

Third, in cases where it has been difficult to model the characteristics of a site or a process on a generic basis, several choices of parameters have been made to understand the range of potential risk results.

It is important to distinguish between the type of uncertainty included in the generic analysis reported here and the uncertainties that would remain with real sites when they are characterized and modeled in connection with the decision on where to put a repository. Many of the uncertainties included here might better be characterized as variabilities. At a real site there might be a wide variation in the property in question. The attempt in these risk assessments to include such variation corresponds then to an uncertainty in the final results as to how well they characterize the performance of the repository. A real site will include the additional uncertainties associated with data collection, site complexity, and difference of opinion about a specific site's characteristics. Such uncertainties are not within the scope of the work reported here.

The following parts of this section assess several of the uncertainties in this risk assessment by examining the sensitivity of the results to many of the assumptions used in the various repository models.

8.11.1 Sensitivity to Waste Package Parameters

The analyses summarized in this section investigated how the population risks and individual exposures described in Section 8.10 vary with different assumptions about waste package lifetime and waste form release rate. Waste package lifetimes of zero and 1000 years were considered for population risks, and lifetimes of 300 and 1000 years were considered for the individual exposures. The waste form release rate was varied from one part in 1000 (10^{-3}) per year to one part in 1,000,000 (10^{-6}) per year.

Figure 8.11-1 displays the results of these analyses for the three different media (basalt, bedded salt, and tuff) being considered for the first repository. Figure 8.11-2 displays similar results for two of the conceptual models used for repositories in granite. As these figures show, variations over the range of waste package lifetimes considered have relatively little effect on the population risks--except for the bedded salt models, where a very short canister lifetime coupled with a rapid release rate allows some of the short-lived fission products to be brought to the land surface by inadvertent intrusion. On the other hand, variations in waste form release rate can cause the projected population risks within 10,000 years to vary by up to three orders of magnitude where releases through ground water flow pathways dominate the analysis.

Figure 8.11-3 shows how the potential individual exposures for normal ground water flow from the basalt model vary for four different assumptions about waste form release rate and two different waste package lifetimes. Increases in package lifetime primarily act to delay the time at which exposures begin to appear. Increases in waste form release rate cause higher initial exposures, which then may decrease with time for the larger release rates as the peak concentration passes the 2-km point of interest.

8.11.2 Sensitivity to Geochemical Parameters

The analyses described in this section indicate how the population risks over 10,000 years for the basalt and bedded salt models vary with different assumptions about the geochemical parameters. For one set of analyses, the geochemical retardation factors were varied to consider the two alternative sets indicated in Table 8.11-1, which are primarily taken from a NAS report (NAS83). For another set of analyses, the solubility limits used for those elements were varied up and down by two orders of magnitude from those used in the base cases of Section 8.10. Figure 8.11-4 shows the results of these variations. The variations had significant effects on the results in only a few cases. Increasing the solubility limits substantially increased the risks for the granite II model and the bedded salt models, while lowering the geochemical retardatioin significantly increased the risks predicted for the tuff model. Lowering the solubility limits or increasing geochemical retardation generally did not substantially reduce the risks projected for the reference cases.

8.11.3 Sensitivity to Americium Solubility

One important change from the analyses supporting the proposed rule concerned the solubility limit assumed for the americium radioisotopes. Because of uncertainty regarding this parameter, the Agency chose a very conservative upper bound of 50 parts per million (ppm) for its earlier analyses. This was one of the parameters that the SAB suggested be reexamined, and more recent information supports the much lower choices

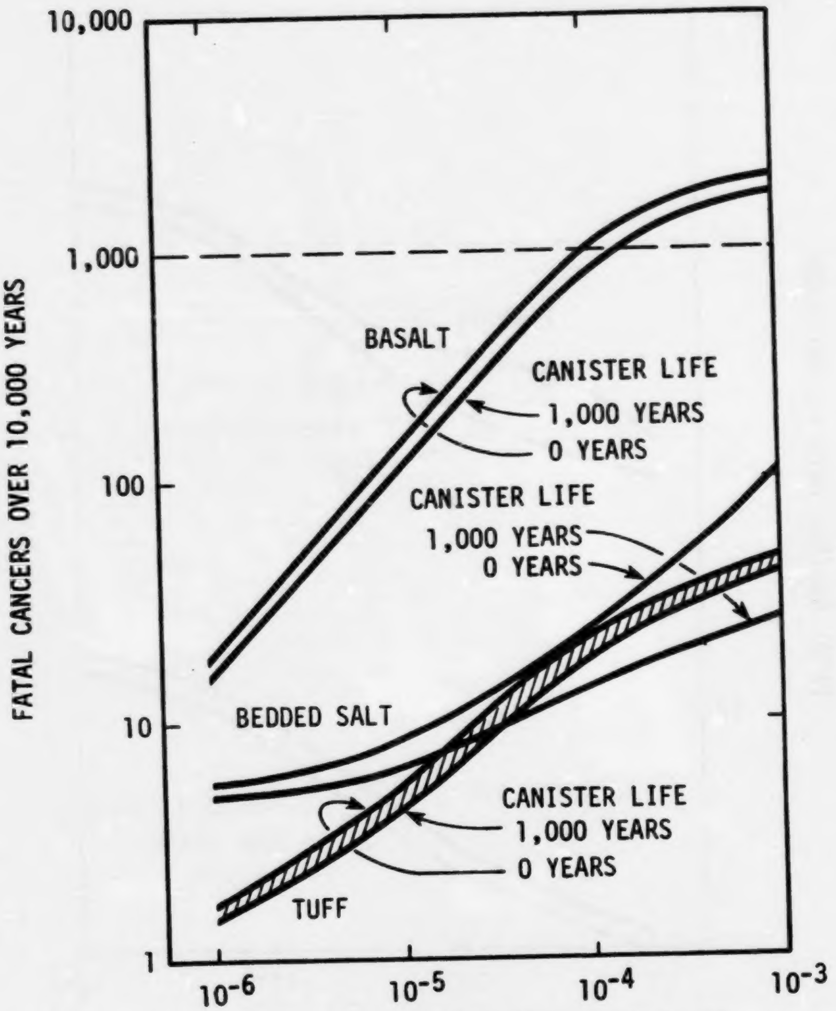


Figure 8.11-1. The effect of canister life and waste form leach rate on population risks for three potentially suitable repository media.

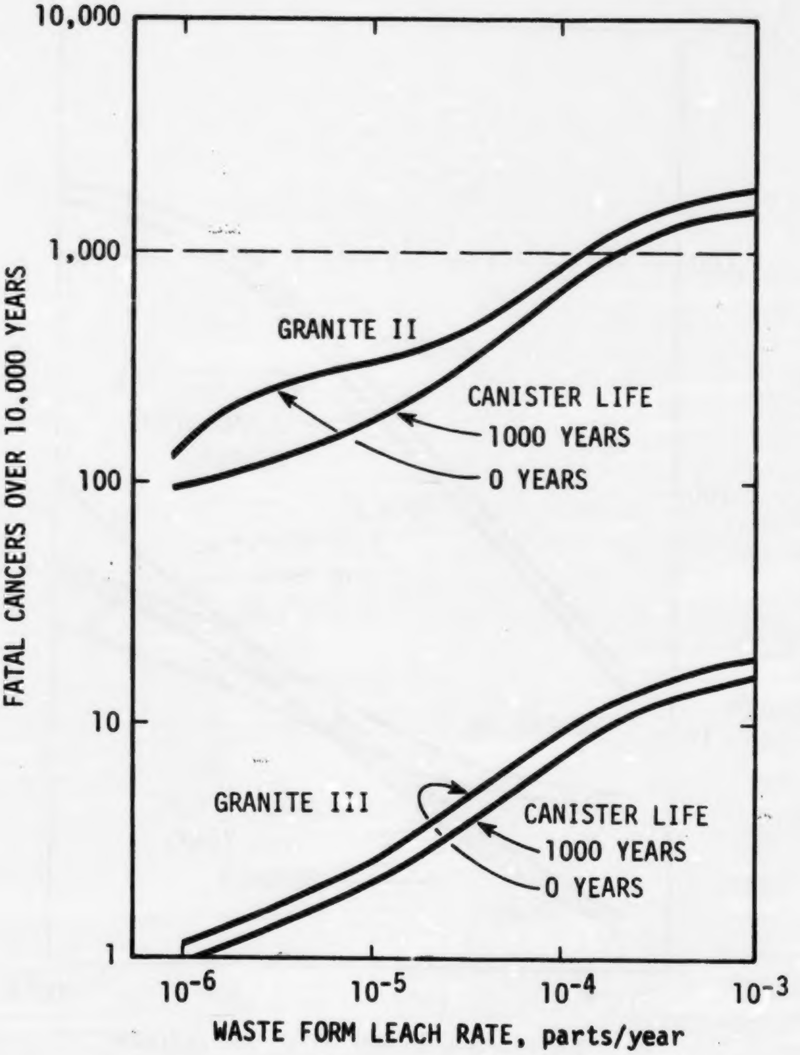


Figure 8.11-2. The effect of canister life and waste form leach rate on estimated population risks for repositories in granitic formations.

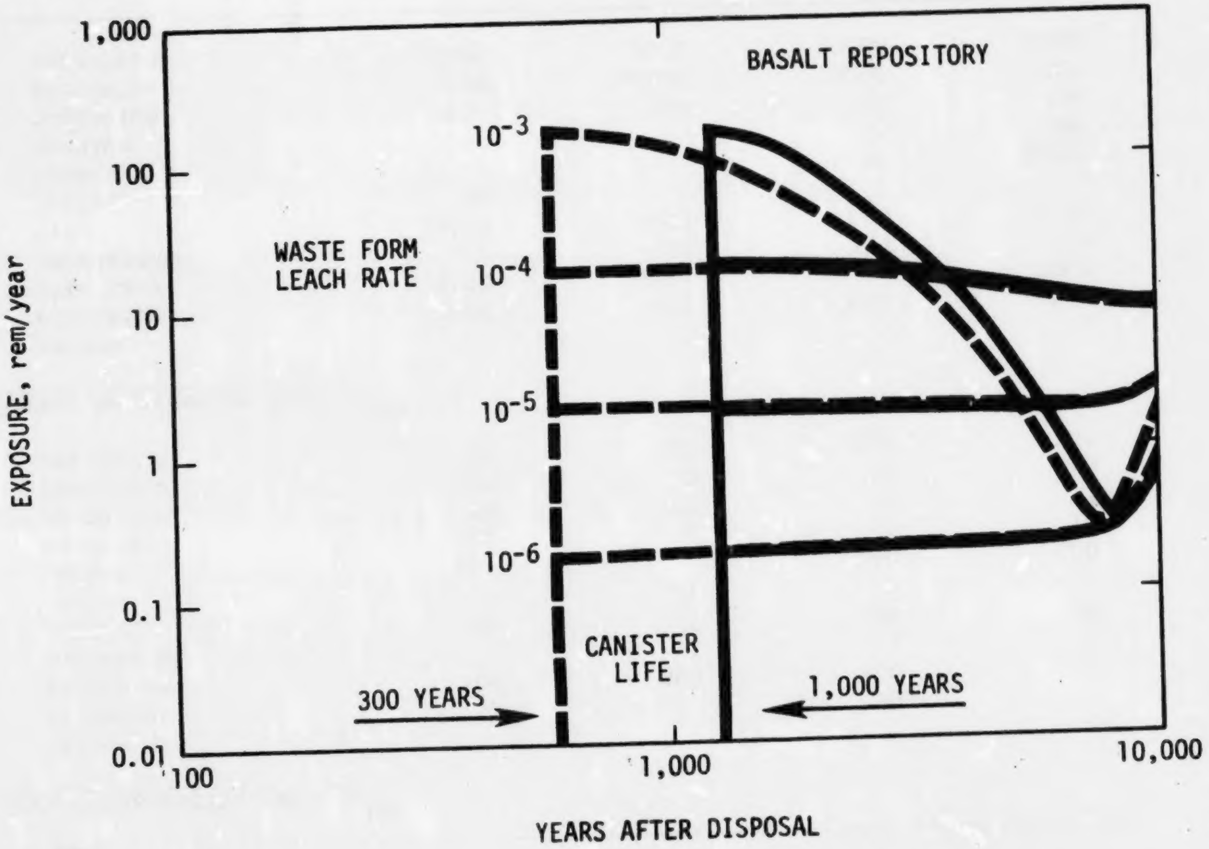


Figure 8.11-3. Effect of canister life and waste form leach rate on radiation exposures from drinking ground water at 2 kilometers from repository.

Table 8.11-1. Alternative geochemical parameters considered in risk assessment

Parameter	Basalt	Bedded Salt	Tuff	Granite
Lower Retardation Factors[a]				
carbon	1	1	1	1
strontium	50	1	20	10
zirconium	500	300	500	500
technetium	1	1	1	1
tin	100	10	200	100
iodine	1	1	1	1
cesium	100	1	60	100
uranium	20	10	5	10
neptunium	10	10	10	10
plutonium	100	10	50	10
americium	60	300	300	500
Higher Retardation Factors[a]				
carbon	1	1	1	1
strontium	2000	100	10000	2000
zirconium	5000	1000	5000	5000
technetium	100	20	100	40
tin	5000	1000	5000	5000
iodine	50	1	1	1
cesium	10000	2000	10000	10000
uranium	1000	60	200	500
neptunium	500	300	500	500
plutonium	5000	10000	5000	5000
americium	10000	3000	10000	10000

[a] For use with all isotopes of radionuclides listed.

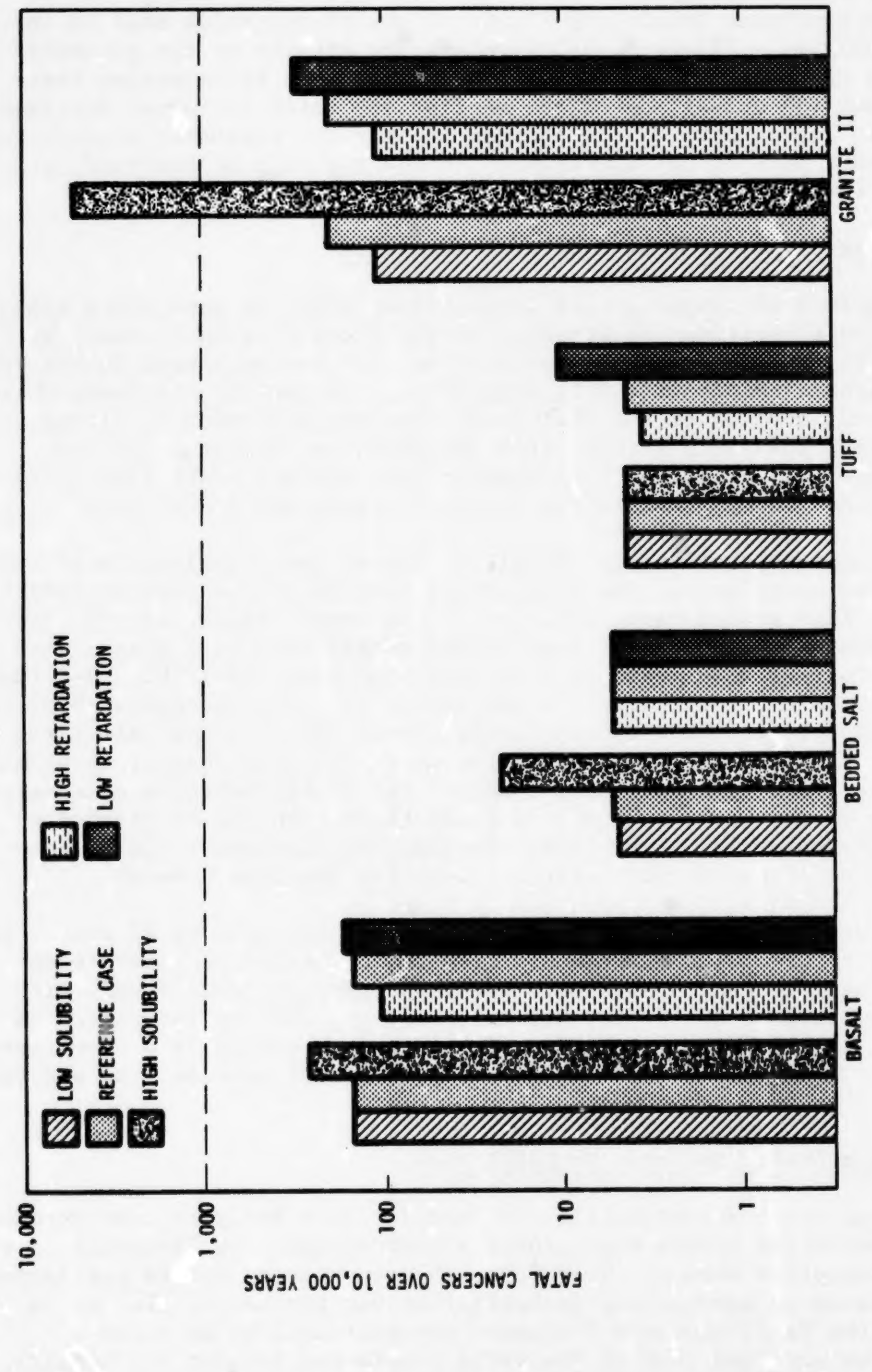


Figure 8.11-4. Effect of geochemical parameters on population risks for different geologic media.

described in Section 8.6 (which vary for the different geologic media). The analyses described in this section indicate the importance of this change. The base case models for basalt and bedded salt and one of the conceptual models for granite were reanalyzed; the only change was to return the americium solubility limits to the 50-ppm value used in the earlier analyses. Figure 8.11-5 displays the effects on the projected population risks over 10,000 years. As can be seen by comparing these results with the comparison of the earlier and later technical analyses included in Figure 8.10-2, the changes in this one parameter account for a significant part of the differences in the two sets of analyses, particularly for the bedded salt models.

8.11.4 Sensitivity to Ground Water Travel Time

The effect of longer ground water travel times on population risks was examined by setting the boundary to the accessible environment at 5 and 10 km in addition to the value of 2 km that has been used in most of these analyses. Since this will only affect the results for cases where a significant portion of the risk comes from release modes involving ground water flow, this section only considers the basalt model and granite model II. Figure 8.11-6 demonstrates how the risks from these two models are changed due to the longer distance and travel time.

The negligible effect for basalt is due to the relationship of the aquifer flow velocity and the retardation factors of the various radionuclides. This ground water velocity in the upper aquifer of the basalt model is about 9.4 meters per year, which corresponds to a travel time of 210 years to 2 km, 530 years to 5 km, and 1060 years to 10 km. Retardation factors are 1 for iodine-129 and carbon-14, 5 for technetium-99, and at least 50 for all other radionuclides. With these values, all three of the fast moving radionuclides reach the accessible environment throughout most of the 10,000-year period for any of the three distances considered, while none of the other radionuclides can reach even the shortest distance in 10,000 years. Therefore, changing the distance through the range of 2 to 10 km has very little effect for the basalt model.

Somewhat more of an effect can be seen for the granite II model, for here the ground water flow velocity is about 80 meters per year in the overlying aquifer (a travel time of only 25 years to cover 2 km), with retardation factors of 100 for neptunium-237 and 200 for the plutonium isotopes. In this case, neptunium and plutonium contribute a significant fraction of the risks for a 2-km distance, but much less at 5 km and none at 10 km.

8.11.5 Sensitivity to Event Probabilities

Most of the event probabilities used in these analyses were intended to be conservative values that probably overestimate the frequency of the various disruptive events. Therefore, the sensitivity of the population risk estimates to lower event probabilities was evaluated. For these analyses, the fault movement frequency was decreased by an order of magnitude or more for each of the media considered (except for basalt).

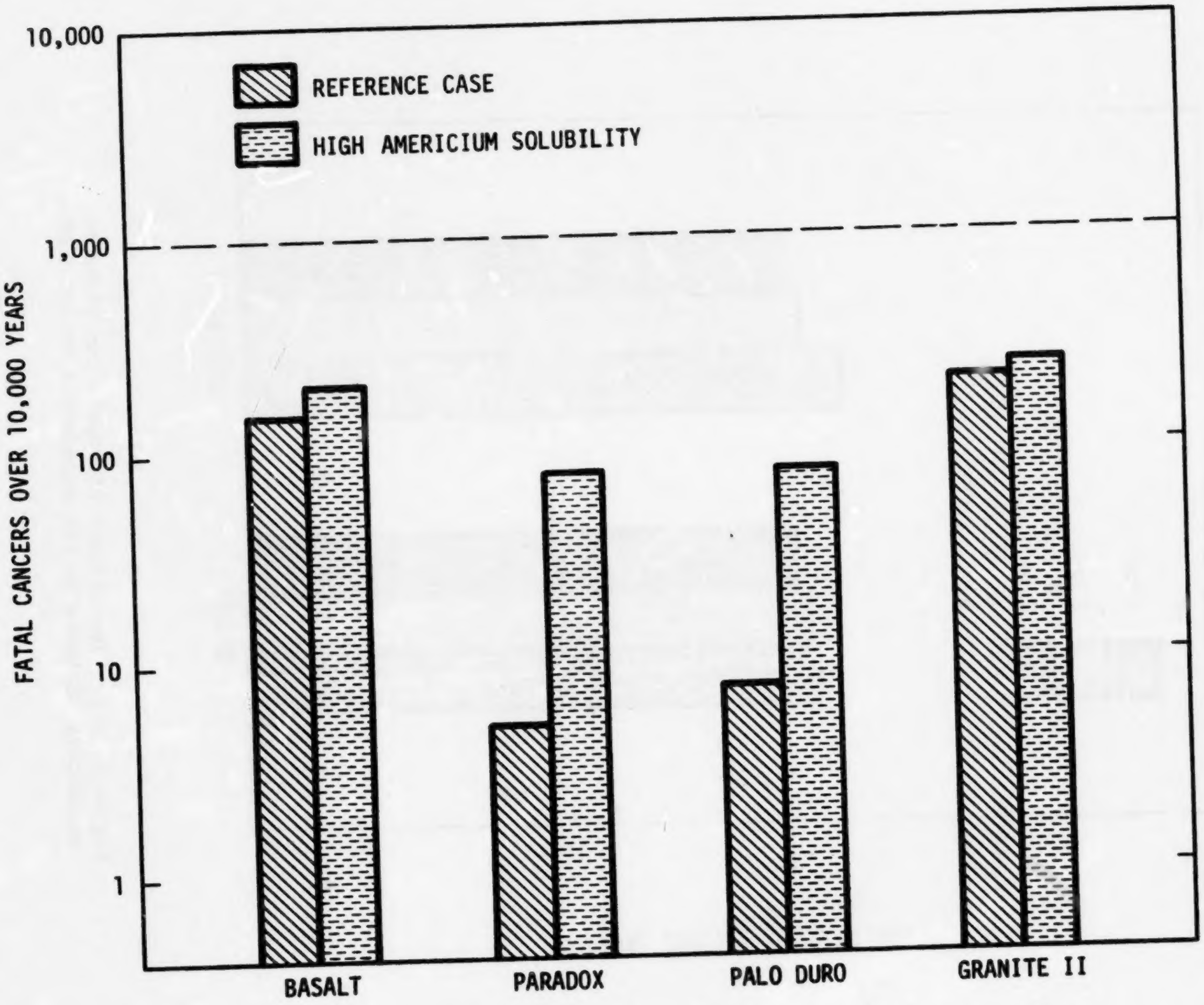


Figure 8.11-5. Effect of solubility limits on population risks for americium in different geologic media.

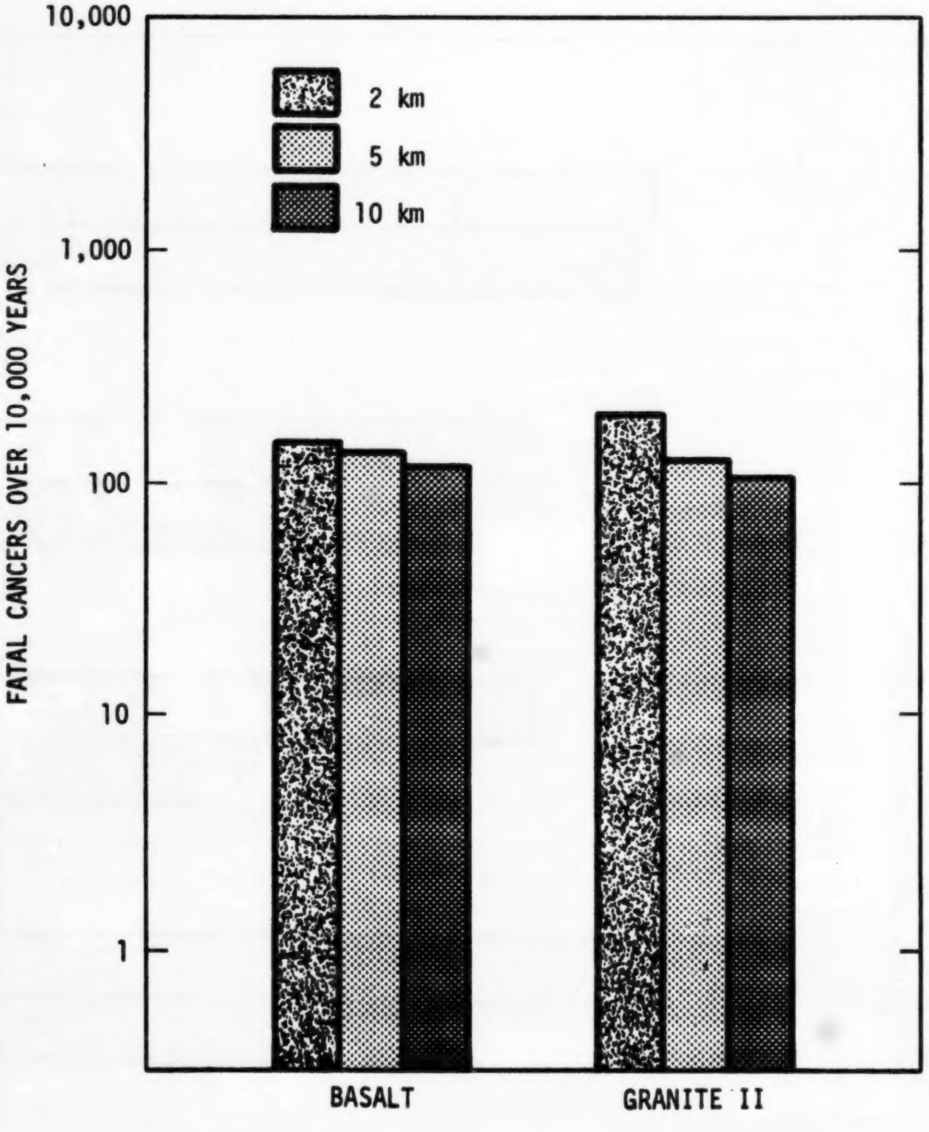


Figure 8.11-6. Sensitivity of population risks to repository distance to the accessible environment.

In addition, the inadvertent human intrusion frequency was decreased by a factor of two, and no intrusions were assumed to occur sooner than 500 years after disposal. Figure 8.11-7 displays the results of these changes for the models in basalt, bedded salt, tuff, and granite. The variations in the population risks in response to these changes are relatively modest, indicating that the risk estimates are not highly sensitive to the values used for the frequency of disruptive events. The most significant reduction in risks with lower probabilities occurred for the tuff model because of the dominance of risk from faulting in the projections for this media.

8.11.6 Sensitivity of Bedded Salt Models to Host Rock Permeability

The models used so far for repositories in bedded salt assume that the salt formation itself is essentially impermeable unless it is disturbed. Therefore, all releases occur either as a result of disruptive events or because of ground water leakage through shaft or borehole seals (and none of the seal leakage pathways results in predicted releases to the accessible environment within 10,000 years after disposal).

In the analyses described in this section, this assumption of impermeability was relaxed in recognition of the possibility that impurities and irregularities in the salt formations could lead to normal ground water flow through the salt. It was assumed that 5 percent of the repository inventory and volume was subjected to ground water flow through the salt, with this flow zone having a hydraulic conductivity of 10^{-9} cm/s and a porosity of 0.05. The gradients and the remainder of the parameters used were the same as for the two bedded salt base case models.

No results are displayed for these analyses, however, because these changes caused no additional releases for either of the bedded salt models. Therefore, the finding that there are no projected releases due to undisturbed performance of bedded salt repositories appears to hold even if there are small permeable zones of this magnitude through the salt formation.

8.11.7 Sensitivity of Tuff Model to Assumptions About Flow Through the Unsaturated Zone

The tuff model projects no releases for normal ground water flow within 10,000 years after disposal because it takes 10,000 years for water to flow downward from the repository through the unsaturated zone and to the top of the underlying aquifer, based upon the hydrological parameters assumed for the unsaturated zone. One of the unresolved questions regarding this model, however, involves the possibility that flow through the unsaturated zone may be predominately through fracture zones in the rock, which would allow water to reach the underlying aquifer much faster. The analyses in this section address this possibility by increasing the hydraulic conductivity assumed for the unsaturated zone by a factor of 5 and decreasing the effective porosity by a factor of 10, thus decreasing the travel time to the lower aquifer by a factor of 50. Figure 8.11-8 displays the results of this change as a function of waste

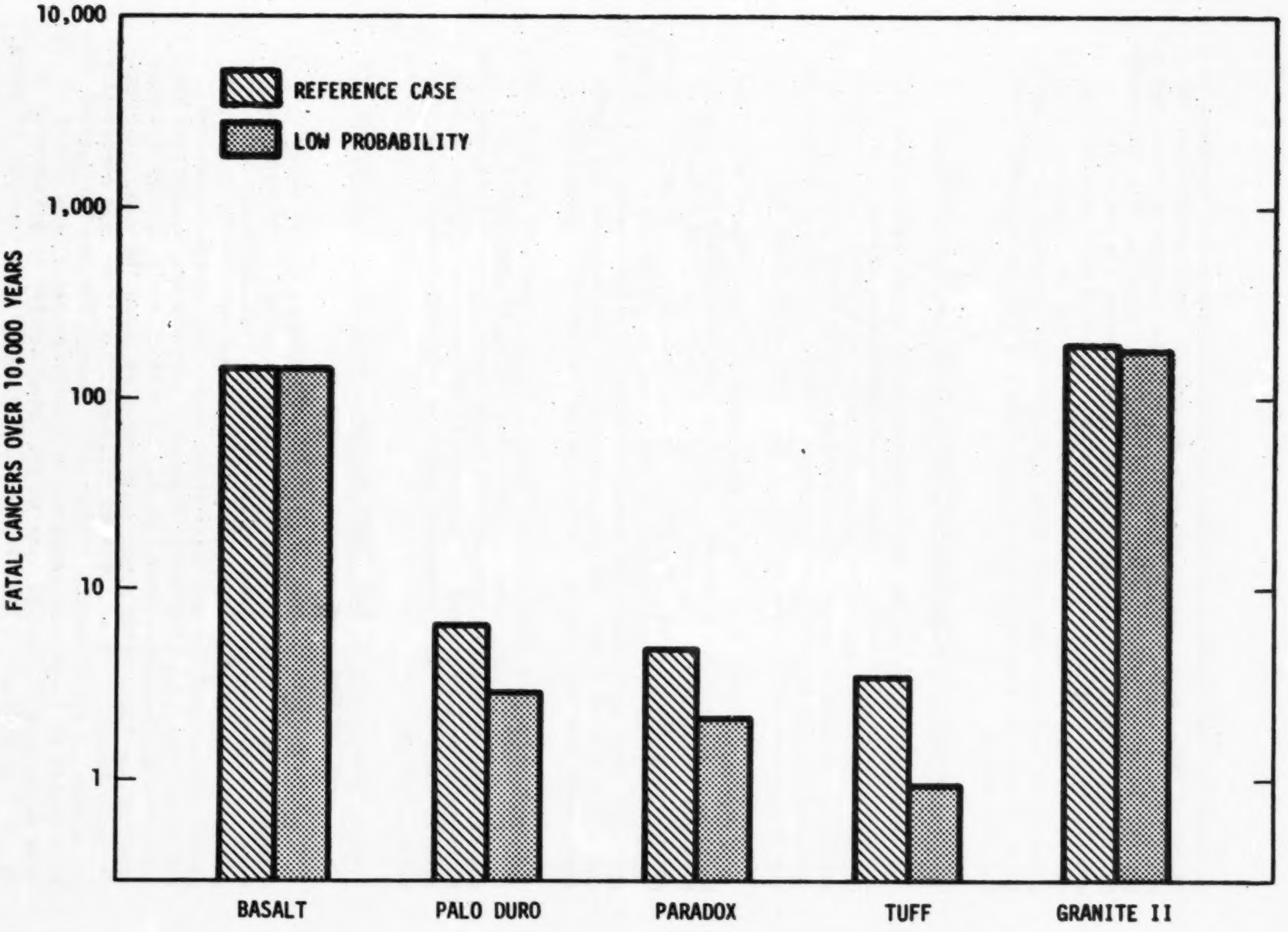


Figure 8.11-7. Sensitivity of population risks to event probabilities.

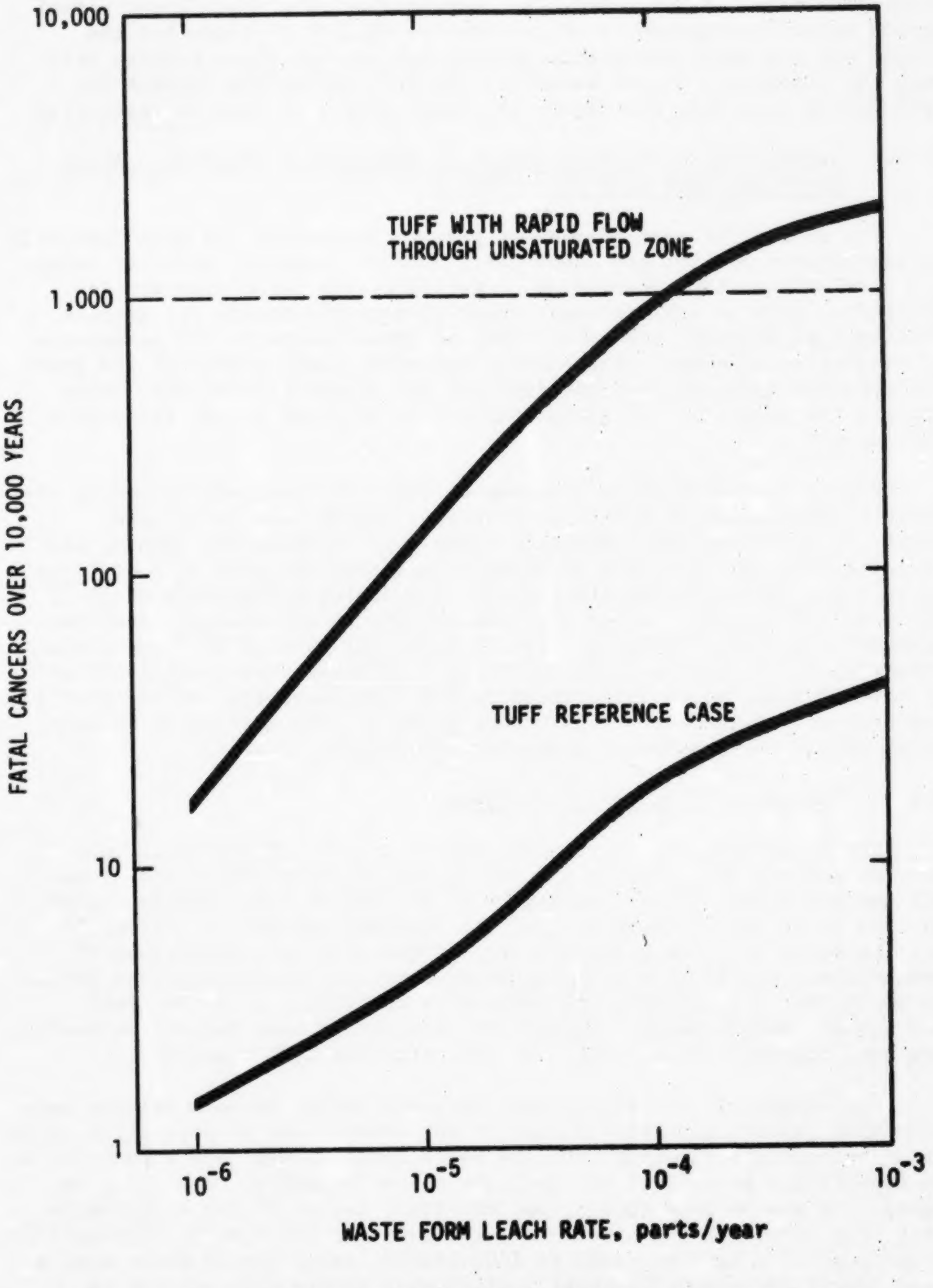


Figure 8.11-8. Sensitivity of population risks to ground water flow in tuff formations.

form leach rate. As expected, the introduction of a significant normal ground water flow pathway produces results similar to those for the basalt and the more pessimistic granite models, and these results indicate the importance of understanding the flow mechanisms through the unsaturated zone when estimating the risks from this type of repository.

8.11.8 Sensitivity of Granite Models to Assumptions About Repository Depth and Host Rock Conductivity

The models for repositories in granite formations are much less well defined than those for the other media because potential sites of interest have not yet been identified. Therefore, the basic analyses described in Section 8.9 included ranges of possible models for granite. This section explores the sensitivity of these models to two parameters of particular interest: the assumed hydraulic conductivity of the granite used for disposal, and the depth of the granite repository, which affects the length of the ground water flow pathways to the accessible environment.

Figure 8.11-9 displays a number of different analyses involving the various granite models and permits several comparisons to be made. First, it shows how the population risks for the idealized granite model (granite III) as a function of waste form leach rate vary with different assumptions about the granite conductivity (using assumptions of 10^{-7}, 10^{-8}, and 10^{-9} cm/s). Since the conductivity in the granite model corresponding to the New England area (granite II) is also 10^{-7} cm/s, the remaining differences between the highest estimates for granite III and granite II involve the differences in the characteristics of the overlying aquifer. Second, the figure also shows how assumptions of greater depth reduce the population risks for the granite II model.

8.11.9 Summary of Sensitivity Analyses

The sensitivity analyses performed in support of the final rule provide several perspectives concerning the relative importance of the various parameters to the projection of population and individual risks, as well as to the achievability of the disposal standards. First, it must be noted that the great majority of the various combinations of assumptions regarding site and engineered barrier characteristics result in population risk projections indicating compliance with the final containment requirements. Second, it is apparent that certain parameters are more important than others for demonstrating such compliance.

With regard to the engineered barriers, the waste form release rate is always appears more significant to the predictions of population risks than the canister lifetime, and the waste form release rate appears to be as significant as many of the geologic characteristics. Thus, a good waste form may be able to overcome uncertainties about the characteristics of a site. On the other hand, the canister lifetime is particularly significant for keeping risks to individuals (using ground water near a repository) to acceptable levels, since even a very good waste form

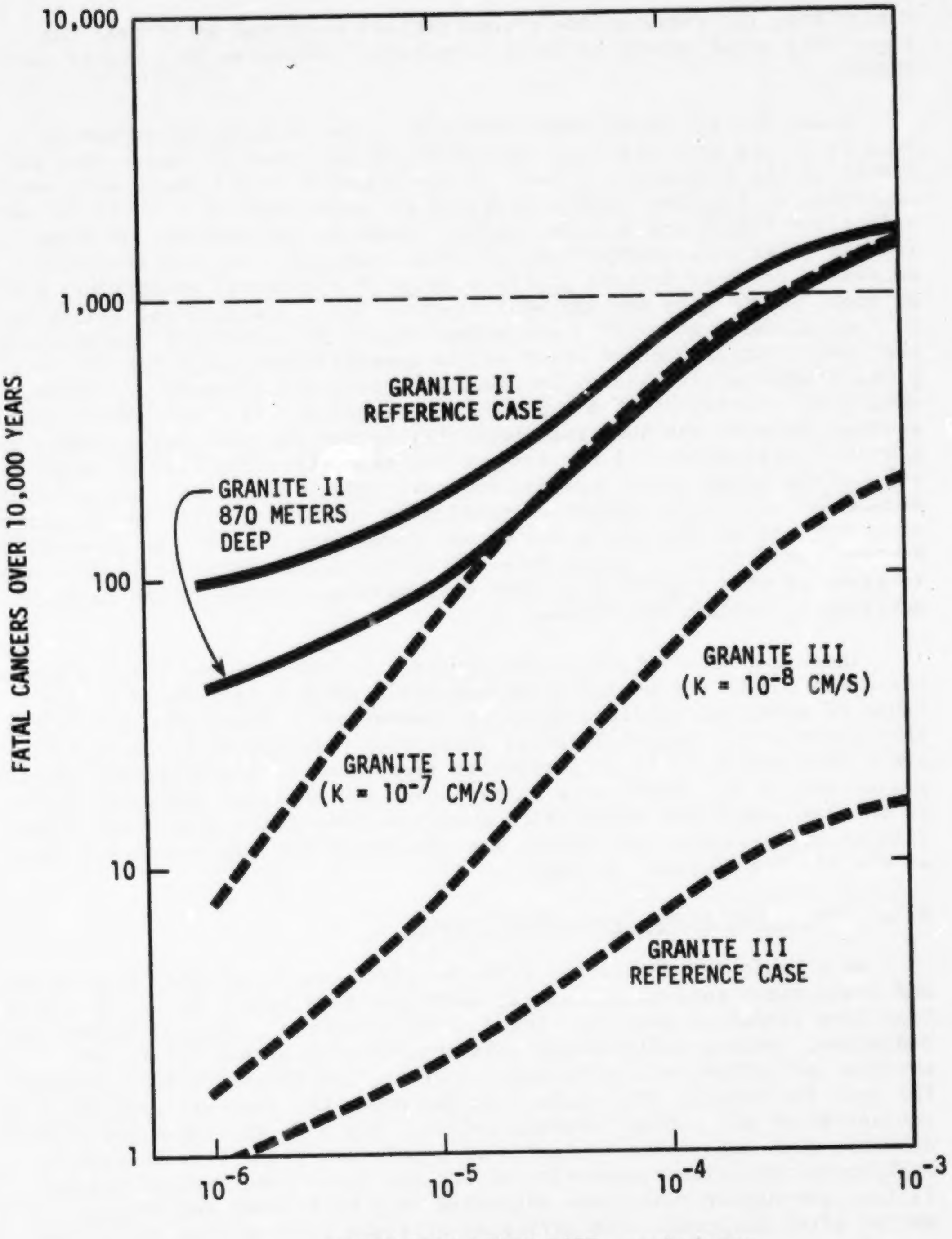


Figure 8.11-9. Sensitivity of population risks to the repository depth and host rock conductivity in granitic formations.

cannot keep the radionuclide concentrations in ground water near the repository small enough to avoid significant exposures to a nearby individual.

Among the geological characteristics, the analyses performed in support of the proposed rule indicated that geochemical retardation and limits of the solubility of many of the elements in the waste were very important to long-term risks, compared to cases where no retardation or solubility limits are assumed (Sm82). However, the analyses performed for the final rule suggest that the risks generally are not unusually sensitive to these factors within a range of reasonable assumptions such as those provided by the NAS WISP report. Thus, assuming the existence of some minimal retardation and modest limits on solubility, determining the exact contribution of these rather uncertain processes may not be of primary importance relative to compliance with the disposal standards. Geological parameters that appeared more important in these sensitivity studies included the host rock permeability for the conceptual models of granite repositories and the assumptions made about the flow of water through the unsaturated zone for the tuff model. Relatively unimportant parameters within the ranges of sensitivty considered were the frequency of occurrence of disruptive events and travel distance to the accessible environment (although travel time and distance are particularly important in terms of when significant risks to individuals using ground water at a particular location may occur).

These analyses reinforce the Agency's belief that the final disposal standards can be successfully implemented. There appear to be a wide range of potential geologic media and repository designs that can reasonably achieve the desired level of protection. Furthermore, those parameters that appear to be of particular importance to the long-term release projections (e.g., waste form release rate and physical characteristics of the host rock) are those which should be relatively well known after site characterization and design and testing of the engineered portions of the planned disposal systems.

8.12 Radiation Risks from Other Sources

As a basis for comparison with the risks associated with high-level and transuranic radioactive waste, estimates have been made of the risks from five radiation sources. Three of these sources, natural background radiation, natural radionuclide concentrations in ground water, and uranium ore bodies, are relatively constant, and the risks will continue far into the future. The risks from the other two sources, generation of nuclear power and nuclear weapons fallout, are more near-term and will decrease with time. The estimates of population risks from natural background radiation, generation of nuclear power, and nuclear weapons fallout are higher than those estimated from high-level radioactive wastes after disposal. The estimates of risks from uranium ore bodies vary over a large range. Some of the estimates of the risks from the disposal of high-level radioactive wastes are slightly above the lower bound of this range and are much less than the higher part of the range.

8.12.1 Risks Associated with Natural Background Radiation

Humans are exposed to natural background radiation from cosmic rays, from naturally occurring radionuclides in the earth, and from naturally occurring radionuclides in the body, primarily potassium-40. Cosmic radiation originates primarily in galactic and solar processes. Naturally occurring radionuclides in the earth's crust are chiefly composed of members of the uranium-238 series, members of the thorium-232 series, and potassium-40. There are approximately 2.7 grams of uranium-238, 9.6 grams of thorium-232, and 20,900 grams of potassium per metric ton of rock in the earth's crust (Fo71). Rock in the earth's crust contains about 1.4×10^{-5} curies of alpha activity per metric ton and about 2.7×10^{-5} curies of beta activity.

The United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) has estimated that the average annual radiation dose from external exposure to natural sources in "normal" areas of the world includes 28 millirads of cosmic radiation and 32 millirads of terrestrial radiation (UNSCEAR77). The UNSCEAR committee stated that the annual dose from external and internal radiation was 92 millirads to red marrow, 78 to gonads, and 110 to lungs. The corresponding annual dose equivalent rates, with a quality factor of 10 for the alpha and neutron components, are 109, 86, and 425 mrems, respectively. The difference among organs is due to differing radionuclide contents in the organs.

People in the United States annually receive from about 50 to 195 mrems natural background radiation dose equivalent from cosmic, terrestrial, and internal radiation. The highest annual dose equivalents received in large urban areas are about 130 mrems in Denver, Colorado, and about 115 mrems in Albuquerque, New Mexico. The highest annual average for a state is about 115 mrem in Colorado. Table 8.12-1 gives the distribution of the dose received by the population. It shows that about 60 percent of the United States population receives an annual dose ranging from 65 to 90 mrems; 20 percent more than 90 mrems; and 5 percent more than 105 mrems.

Information on the variability of the dose equivalent received by people in the United States has been published by Oakley (Oa72) and revised by Bogen (Bo80). Cosmic radiation annual dose equivalent in the United States varies from 29 mrems at sea level to about 125 mrems at 3200 meters (10,500 feet), the altitude of Leadville, Colorado (NCRP75). The terrestrial component dose equivalent from radionuclides in the earth can be characterized as low (annual average of 23 mrems) for the Atlantic and Gulf coastal plain, moderate (annual average of 46 mrems) for most of the rest of the United States, and high (annual average of 90 mrems) for a small region in the Colorado Front Range.

Table 8.12-1. Distribution of natural radiation annual dose equivalents (Terrestrial, cosmic, and internal)

Dose equivalent[(a)] (mrem)	Percentage of U.S. Population receiving more than stated dose
50	100
65	80
75	50
90	20
105	5
120	1

(a) Annual dose equivalent includes 25 millirems from internal radiation. Values have been rounded to the nearest 5 mrem.

It is estimated that an excess of about 280 fatal cancers per million person-rem can be expected (see Chapter 7). This means that an increase of 1 mrem/yr in natural background radiation (about 1 percent) to everyone in the present U.S. population would result in about 60 excess fatal cancers per year. A 1-mrem/yr increase in radiation exposure to 5 million people (a population that might be exposed to releases from a large high-level radioactive waste repository) would result in about one additional fatal cancer per year.

Risks associated with natural background radiation are similar to those from radioactive wastes; both produce the same kinds of health effects and both persist for long periods of time. The risk from natural background radiation is much larger than that projected for releases from a high-level radioactive waste repository. However, in general, the variability of this risk does not seem to greatly influence the choices people make in selecting a place to live.

8.12.2 Risks Associated with Natural Radionuclide Concentrations in Ground Waters

One part of the human exposure to background radiation comes from naturally occuring radionuclides in the earth that, in turn, are taken up by ground water. Radium is the most important of these naturally occurring materials likely to occur in public water supply systems. Uranium is also found in ground waters due to its natural occurrence. Thus, it is expected that some drinking water sources will contain concentrations of uranium (Co83,84).

Surveys of radionuclides in ground water systems indicate a United States range of: 0.1 to 50 picocuries/liter (pCi/l) for radium-226 (with some isolated sources exceeding 100 or more pCi/l); 0 to 74 pCi/l for gross alpha concentrations (although most of the United States gross alpha concentrations are below 3 pCi/l); and 0 to 650 pCi/l for total uranium. Elevated radium-226 concentrations are seen along the Atlantic coastal region (North and South Carolina, Georgia, and Florida) and the Midwest (Illinois, Wisconsin, Minnesota, and Kansas). Low levels, or almost none, are found in treated drinking waters in the western states. In general, only those locations with elevated uranium concentrations show elevated alpha concentration levels, and these are primarily in the Rocky Mountain region (New Mexico, Colorado, Wyoming, and Montana) and in Maine and Pennsylvania in the east (Co83, Dr81).

Of the nearly 60,000 community water supplies in the United States, about 80 percent use ground water sources, and over 90 percent of the ground water supplies serve less than 3,300 people. In general, radium in drinking water is a problem for small water supply systems, with an estimated 500 supplies exceeding 5 pCi/l (Co84). It is estimated that less than 650 community water supplies exceed 20 pCi/l of uranium (Co83, Dr81).

The Agency's Interim Drinking Water Regulations (40 CFR Part 141) for radionuclides limit the contamination levels for radium-226 and radium-228 to no more than 5 pCi/l and limit gross alpha particle activity (including radium, but excluding radon and uranium) to 15 pCi/l (EPA76). Risk estimates derived from the BEIR Report indicate that continuous consumption of drinking water containing radium-226 or radium-228 at the maximum contaiminant level of 5 pCi/l may cause between 0.7 and 3 cancers per year per million exposed persons. Almost all of these cancers would be expected to be fatal (EPA76).

8.12.3 Risks from Uranium Ore Bodies

A comparison of the risks from a high-level waste repository with those from an undisturbed uranium ore body offers another useful perspective. In this assessment, it is assumed that uranium dissolved by the ground water eventually reaches a stream (Wi80). As the uranium moves along the aquifer, radium-226 and other daughters are formed. An ore body containing 620,000 tons of uranium oxide would have to be mined to produce the high-level radioactive wastes contained in the model repository. An estimate of the risks presented by this ore body was performed using the same generic environmental model described in Chapter 7 of this report (Sm85).

The risks from uranium ore bodies cover a wide range, depending on the ability of uranium and its decay products (particularly radium-226) to leave the ore body and reach people. To estimate a minimum impact, a uranium concentration in ground water at the low end of the reported range was selected (Fi55). The Agency's orginal assessment in Wi80 has been updated for the changes made in the assessment of the effects of releasing a curie of radium-226 to the environment in Sm85 (a reduction of about a factor of 30). This minimum impact is now estimated to be about 10 excess fatal cancers over 10,000 years for the model generic ore body. Based on pre-operational data for three actual uranium mines reported in Wi80, the maximum impact from the generic ore body is estimated to be about 100,000 excess cancers over 10,000 years. These estimates based on data from uranium mines are likely to be high because the reported concentrations in the ground water include some measurements from the oxidizing uranium-rich ground water. Therefore, the risks to future generations from an amount of uranium ore equivalent to that which would be used to produce 100,000 metric tons of spent fuel would appear to range from 10 to 100,000 fatal cancers over 10,000 years.

8.12.4 Risks from Nuclear Power Generation

Ellett and Richardson estimated that exposure to the radiation incident to the generation of 1 gigawatt-year of electrical energy could result in an average of about 1.2 fatal cancers* in the first 100 years (E177).

More recent information reduces this estimate to 0.8 fatal cancers. Operation of the 50 gigawatts of nuclear power generation available in 1977 at 60 percent of capacity could cause about 24 fatal cancers per year (ADL79). To the extent that radionuclides such as carbon-14, thorium-230, and radium-226 and its daughters persist beyond 100 years, this estimate may be low.

8.12.5 Risks from Nuclear Weapons Fallout

Fallout from atmospheric testing of nuclear weapons has added to the radioactivity of air, water, food, and residual radionuclides (primarily tritium, strontium-90, cesium-137, carbon-14, and plutonium) and is a source of continual radiation exposure. UNSCEAR has estimated the dose commitments to various organs of an average individual in the northern temperate zone from nuclear weapons testing through 1975 (UNSCEAR77). When converted to millirems, the dose commitments are 150 millirems to gonads, 260 to bone marrow, 300 to bone linings, and 270 to lung. Almost all the dose commitment is due to relatively short-lived (30 years or less) nuclides, because doses for carbon-14 were calculated only to the year 2000.

The number of projected fatal cancers in the United States population from fallout was estimated by multiplying the average dose commitments by a population of 200 million and by the appropriate risk factors. It is assumed that the soft tissue dose is equal to the gonadal dose. Thus, the whole body risk factor was reduced from 200 per million person-rem to 110, to avoid double counting the fatal cancers from the listed organs. The expected harm from nuclear weapons testing in the United States is then about 3300 fatal cancers from soft tissue irradiation, 2080 from bone marrow irradiation, 600 from bone lining irradiation, and 2160 from lung irradiation, for a total of 8140 fatal cancers

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* The estimated number of fatal cancers represents a probability of incidence in the entire exposed population.

over the life of the radionuclides involved. If this number is averaged over the half-lives of cesium-137 and strontium-90, the largest contributors to the dose commitment, the result is an average of about 190 fatal cancers per year over about 43 years.

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APPENDIX A

A DESCRIPTION OF THE RADRISK AND CAIRD COMPUTER CODES USED BY EPA TO ASSESS DOSES AND RISKS FROM RADIATION EXPOSURE

A.1 Introduction

This appendix provides a brief overview of the RADRISK (Du80) and CAIRD (Co78) computer codes used by the Environmental Protection Agency to assess the health risk from radiation exposures. It describes how the basic dose calculations are performed and describes the mechanics of the life table implementation of the risk estimates derived in Chapter 6.

A.2 Overview of the EPA Analysis

RADRISK, the computer code used to calculate dose and risk, calculates the radiation dose and risk resulting from an annual unit intake of a given radionuclide or the risk resulting from external exposure to a unit concentration of radionuclide in air or on ground surface (Du80,84, Su81). Since both dose and risk models are linear, the unit dose and risk results can then be scaled to reflect the exposure associated with a specific source.

As outlined in Chapter 5, estimates of the annual dose rate to organs and tissues of interest are calculated by using, primarily, models recommended by the International Commission on Radiological Protection (ICRP79,80). Because EPA usually considers lifetime exposures to a general population, these dose rates are used in conjunction with a life table analysis of the increased risk of cancer resulting from radiation (Co78). This analysis, described below, takes account of competing risks and the age of the population at risk.

A.3 Dose Rates from Internal Exposures

Internal exposures occur when radioactive material is inhaled or ingested. The RADRISK code implements contemporary dosimetric models to estimate the dose rates at various times to specified reference organs in the body from inhaled or ingested radionuclides. The dosimetric methods in RADRISK are adapted from those of the INREM-II code which is based on models recommended by the International Commission on Radiological Protection (Ki78, ICRP79). The principal qualitative difference is that RADRISK computes dose rates to specified organs separately for high- and low-linear energy transfer (LET) radiations, while INREM-II calculates the committed dose equivalent to specified organs. The time-dependent dose rates are used in the life table calculations of RADRISK.

In RADRISK, the direct intake of each radionuclide is treated separately. For decay chains, the ingrowth and dynamics of decay products (daughters) in the body after intake of a parent radionuclide are considered explicitly in the calculation of dose rate. The decay product contributions to the dose rate are included in the dose calculations, based on the metabolic properties of the element and the organ in which they occur.

The dose rate $\dot{D}_i(X;t)$ to target organ X at time t due to radionuclide i ($1 \le i \le N$) residing in organs Y_1, Y_2, ..., Y_m is a measure of the energy deposited annually in a given mass of tissue as a result of radioactive decay, and is computed as:

$$\dot{D}_i(X;t) = \sum_{k=1}^{m} \dot{D}_i(X \leftarrow Y_k;t) \qquad \text{(A-1)}$$

where

$$\dot{D}_i(X \leftarrow Y_k;t) = S_i(X \leftarrow Y_k)A_{ik}(t) \qquad \text{(A-2)}$$

$A_{ik}(t)$ = activity of radionuclide i in organ Y_k at time t measured from the initial intake of radionuclide i into the body,

$S_i(X \leftarrow Y_k)$ = average dose rate to target organ X per unit activity of the radionuclide i uniformly distributed in source organ Y_k (Sn74, Du80).

The summation is taken over all source organs Y. Implicit in the definitions is the assumption of uniform distribution of activity of radionuclide i in each source organ, as is the assumption of averaging the dose rate over the mass of the target organ. Although estimates of dose to an organ include contributions from activity distributed throughout the body (for penetrating radiations), activity within that organ generally contributes the principal component of dose [i.e., $\dot{D}_i(X \leftarrow X;t)$ is the principal component of $\dot{D}_i(X;t)$].

The time rate of change of activity in the body is modeled by a system of ordinary differential equations, with each equation describing the rate of change of activity in a conceptual compartment of the body. For radionuclides that are part of a decay series, there may be formation of radioactive daughters in a given compartment that have different chemical and physical properties from those of the parent. Unlike the models given in ICRP80, the specific metabolic properties of the daughter are modeled when they differ from those of the parent. This refinement is under active consideration by ICRP experts. In almost all cases, doses to soft tissues calculated on this basis differ only slightly, if at all, from ICRP80 dose estimates, but the difference is large for some radionuclides when the parent is incorporated into bone, as in lead-210. For this radionuclide the ICRP80 model has been used without any modifications.

A schematic representation of radionuclide movement in the body was shown in Chapter 5. Except for radon daughters, which are considered separately, inhaled radionuclides are assumed to be originally deposited in the lungs (distributed among the nasopharyngeal, tracheobronchial, and pulmonary regions), and ingested radionuclides are originally deposited in the stomach. From the lungs, radionuclides may be absorbed by the bloodstream or migrate to the stomach. Radionuclides in the stomach may proceed through the small intestine, upper large intestine, and lower large intestine; radionuclides may be absorbed by the bloodstream from any of these four segments, although only absorption from the small intestine is considered in this study.

The activity, $A_{ik}(t)$, of radionuclide i in organ k may be divided among several "pools" or "compartments," denoted here by the subscript ℓ. Each differential equation describing the rate of change of activity within a compartment is a special case of the equation:

$$\dot{A}_{i\ell k} = -(\lambda_i^R + \lambda_{i\ell k}^B)A_{i\ell k} + c_{i\ell k}(\lambda_i^R \sum_{j=1}^{i-1} B_{ij} \sum_{r=1}^{L_{jk}} A_{jr} + P_{ik}), \quad \ell=1,\ldots, L_{ik} \tag{A-3}$$

where

$\dot{A}_{i\ell k}$ = activity of radionuclide i in compartment ℓ of organ k,

L_{jk} = number of exponential terms in the retention function for radionuclide j (j=1 to i-1) in organ k,

B_{ij} = branching ratio of radionuclide j to radionuclide i,

λ_i^R = rate coefficient (time^{-1}) for radiological decay of radionuclide i,

$\lambda_{i\ell k}^B$ = rate coefficient (time^{-1}) for biological removal of radionuclide i from compartment ℓ of organ k,

$c_{i\ell k}$ = fractional coefficient for radionuclide i in the ℓ-th compartment of organ k,

P_{ik} = inflow rate of radionuclide i into organ k.

If the inflow rate P_{ik} remains constant, the equations may be solved explicitly for $A_{ik}(t)$ as described by Killough, Dunning, and Pleasant (Ki78). In many cases the inflow into a compartment will not be a constant rate over a long period of time. To handle this problem, the time interval over which solution of the activity equation is desired (e.g., 110 years) is divided into 1-year subintervals. The inflow rate on each subinterval is then taken to be that constant value which would yield the total activity flowing out of the preceding compartment(s) during the same subinterval.

The model used in RADRISK for particulate deposition and retention in the respiratory tract is the ICRP task group lung model (Mo66, ICRP72).

In this model, shown in Chapter 5, there are four major regions: the nasopharyngeal, tracheobronchial, pulmonary, and lymphatic tissues. A fraction of the inhaled radioactive material is initially deposited in each of the nasopharyngeal, tracheobronchial, and pulmonary regions. The material is then cleared (removed) from the lung to the blood and the gastrointestinal tract, also as shown in Chapter 5. Deposition and clearance of inspired particulates in the lung are controlled by the particle size and solubility classification.

The size distribution of the particles is specified by the activity median aerodynamic diameter (AMAD); where no AMAD is known, a value of 1.0 micron is assumed. The model employs three solubility classes, based on the chemical properties of the radionuclide; classes D, W, and Y correspond to rapid (days), intermediate (weeks), and slow (years) clearance, respectively, of material deposited in the respiratory passages. Inhaled nonreactive, i.e., noble, gases are handled as a special case.

Movement of activity through the gastrointestinal (GI) tract is simulated with a catenary model, consisting of four segments: stomach, small intestine, upper large intestine, and lower large intestine. Exponential outflow of activity from each segment into the next or out of the system is assumed. Outflow rate constants are calculated from the transit times of Eve (Ev66). Although absorption may occur from any combination of the four segments, only activity absorbed into the blood from the small intestine is normally considered; the fractional absorption from the small intestine into the blood is traditionally denoted f_1.

Activity absorbed by the blood from the GI or respiratory tract is assumed to be distributed immediately to systemic organs. The distribution of activity to these organs is specified by fractional uptake coefficients. The list of organs in which activity is explicitly distributed (termed source organs) is element-dependent, and may include such organs as bone or liver where sufficient metabolic data are available. This list is complemented by an additional source region denoted as OTHER, which accounts for that systemic activity not distributed among the explicit source organs; uniform distribution of this remaining activity within OTHER is assumed.

Radioactive material that enters an organ may be removed by both radioactive decay and biological removal processes. For each source organ, the fraction of the initial activity remaining at any time after intake is described by a retention function consisting of one or more exponentially decaying terms.

The metabolic models and parameters employed in the present study have been described by Sullivan et al. (Su81). In most cases, the models are similar or identical to those recently recommended by the ICRP (ICRP79,80,81). However, some differences in model parameters do exist for some radionuclides (Su81). In particular, parameter values that are thought to be more representative of metabolism following low-level environmental exposures, rather than occupational exposures, have been used in this analysis [e.g., f_1=0.2 for uranium in the environment (ICRP79, NAS83)]. For transuranic isotopes, metabolic parameters from EPA77, related comments from EPA78 and from the National Radiological Protection Board (Ha82), have been used rather than those from ICRP80. These parameters are listed in Table A.3-1.

The EPA values were recommended by U.S. experts on transuranic element matabolism at Battelle Pacific Northwest Laboratory (EPA78). The recently-adopted National Radiation Protection Board f_1 values for transuranics in the general environment are closer to the values proposed by EPA in 1977 than those currently advocated by ICRP for occupational exposures. The larger f_1 values will increase the estimated dose and risk from ingestion of transuranic materials but have little effect on doses following inhalation.

A.4 Dose Rates from External Exposures

Because of the penetrating nature of photons, radioactivity need not be taken into the body to deliver a dose to body organs. Energy absorbed from photons emitted by radionuclides in the air or on the ground surface may also contribute to the overall risk. Natural background radiation is an example of an important external exposure, ordinarily contributing the largest component of dose to people.

Organ dose rates to an individual immersed in contaminated air or standing on a contaminated ground surface are computed by Kocher's DOSFACTOR computer code (Ko81). These calculations assume that the radionuclide concentration is uniform throughout an infinite volume of air or area of ground surface, and that the exposed individual is standing on the ground surface. Only photons penetrate the body sufficiently to deliver a significant dose to internal organs, and only doses from photon radiation are considered in this analysis. Beta radiation is far less penetrating and delivers a dose only to the body surface; because skin is not a target tissue of concern in this analysis, no consideration of beta contributions to dose is required. Alpha particles have even less penetration ability, and are also excluded from consideration here.

The photon dose rate factor $\dot{D}_i^{\gamma}(X)$ for a given target organ, X, of an individual immersed in contaminated air at any time may be expressed as:

$$\dot{D}_i^{\gamma}(X) = cK_{pm} \frac{1}{\rho_a} \sum_n f_n^{\gamma} E_n^{\gamma} \left[\frac{(\mu/\rho)t}{(\mu/\rho)a} \right]_n G^X \tag{A-4}$$

Table A.3-1. Small Intestine to Blood Transfer Fractions, f_1, for Transuranic Elements

Element	EPA		NRPB		
				Child	
Isotope	Child 0-12 mo	Adult >12 mo	Adult	0-12 mo	0-3 mo
Plutonium-238 and 241					
Oxide form	10^{-2}	10^{-3}	$10^{-5(b)}$	$5\times10^{10-4(b)}$	$10^{-3(b)}$
Nonoxide form	10^{-2}	10^{-3}	5×10^{-4}	5×10^{-3}	10-2
Bio. inc.(a)	5×10^{-2}	5×10^{-3}	5×10^{-4}	5×10^{-4}	10^{-2}
Plutonium-239 and 240					
Oxide form	10^{-3}	10^{-4}	$10^{-5(b)}$	$5\times10^{-4(b)}$	$10^{-3(b)}$
Nonoxide form	12^{-2}	$1-^{-3}$	5×10^{-4}	5×10^{-3}	10^{-2}
Bio. inc.	5×10^{-2}	5×10^{-3}	5×10^{-4}	5×10^{-3}	10^{-2}
Americium					
Oxide form	10^{-2}	10^{-3}	5×10^{-4}	5×10^{-3}	10^{-2}
Nonoxide form	10^{-2}	10^{-3}	5×10^{-4}	5×10^{-3}	10^{-2}
Bio. inc.	5×10^{-2}	5×10^{-3}	5×10^{-4}	5×10^{-3}	10^{-2}
Curium					
Oxide form	10^{-2}	10^{-3}	5×10^{-4}	5×10^{-3}	10^{-2}
Nonoxide form	10^{-2}	10^{-3}	5×10^{-4}	5×10^{-3}	10^{-2}
Bio. inc.	5×10^{-2}	5×10^{-3}	5×10^{-4}	5×10^{-3}	10^{-2}
Neptunium	-	10^{-3}	10^{-3}	5×10^{-3}	10^{-2}

(a) Biologically incorporated form.
(b) Hydroxide form.

Source: EPA77, EPA78, Ha82.
NRPB: National Radiological Protection Board.

where

ρ_a = density of air,

K_{pm} = 0.5, the particle-medium correction factor,

f_n^γ = intensity of n^{th} discrete photon (number/disintegration),

E_n^γ = energy of n^{th} photon,

μ/ρ = photon mass energy absorption coefficient, with subscripts "t" and "a" denoting tissue and air, respectively, for photons of energy E_n,

G^X = ratio of absorbed dose in organ X to absorbed dose at the body surface.

c = unit conversion proportionality contrast.

The terms μ/ρ and G^X are functions of photon energy, E_n^γ.

The photon dose rate factor $\dot{D}_{iz}^\gamma(X)$ to organ X of an individual at a distance z above a unit concentration contaminated ground surface may be computed as:

$$\dot{D}_{iz}^\gamma(X) = 0.5cK_{pm} \sum_n f_n^\gamma E_n^\gamma[(\mu/\rho)_t]n$$

$$x \int_z^\infty 1/r \exp(-\mu_{an} r)dr$$

$$-[C_{an}/(D_{an} - 1)]\exp[(D_{an} - 1)\mu_{an} z]\ G^x \quad \text{(A-5)}$$

where

K_{pm} = 1.0, the particle-material correction factor,

μ_{an} = mass attenuation coefficient for the n^{th} discrete proton,

z = height of reference position above ground surface (taken to be 1 meter in this study).

c = unit conversion proportionality constant.

The coefficients C_{an} and D_{an} are functions of the photon energy. For detailed discussion of the derivation of these equations and a tabulation of dose rate factors for various radionuclides, see Kocher (Ko79, Ko81).

In the analysis here, the dose rate factors described by these equations are scaled to achieve a continuous exposure of 1 pCi/cm^3 for air immersion and 1 pCi/cm^2 for ground surface exposure. Risk estimates for these exposure pathways are based on continuous lifetime exposure to these levels.

A.5 Life Table Analysis to Estimate the Risk of Excess Cancer

Radiation effects can be classified as stochastic or nonstochastic (NAS80, ICRP77). For stochastic effects, the probability of occurrence of the effect, as opposed to the severity, is a function of dose; induction of cancer, for example, is considered a stochastic effect. Nonstochastic effects are those health effects for which the severity of the effect is a function of dose; examples of nonstochastic effects include cell killing, suppression of cell division, cataracts, and nonmalignant skin damage.

At the low levels of radiation exposure attributed to radionuclides in the environment, the principal health detriment is the induction of cancers (solid tumors and leukemia), and the expression, in later generations, of genetic effects. In order to estimate these effects, instantaneous dose rates for each organ at specified times are sent to a subroutine adaptation of CAIRD contained in the RADRISK code. This subroutine uses annual doses derived from the transmitted dose rates to estimate the number of incremental fatalities in the cohort due to radiation-induced cancer in the reference organ. The calculation of incremental fatalities is based on estimated annual incremental risks, computed from annual doses to the organ, together with radiation risk factors such as those given in the 1980 NAS report BEIR-3 (NAS80). Derivation of the risk factors in current use is discussed in Chapter 6.

An important feature of this methodology is the use of actuarial life tables to account for the time dependence of the radiation insult and to allow for competing risks of death in the estimation of risk due to radiation exposure. A life table consists of data describing age-specific mortality rates from all causes of death for a given population. This information is derived from data obtained on actual mortality rates in a real population; mortality data for the U.S. population during the years 1969-1971 are used throughout this study (HEW75).

The use of life tables in studies of risk due to low-level radiation exposure is important because of the time delay inherent in radiation risk. After a radiation dose is received, there is a minimum induction period of several years (latency period) before a cancer is clinically observed. Following the latency period, the probability of occurrence of a cancer during a given year is assumed to be constant for a specified period, called a plateau period. The length of both the latency and plateau periods depends upon the type of cancer.

During or after radiation exposure, a potential cancer victim may experience years of life in which he is continually exposed to risk of death from causes other than incremental radiation exposure. Hence, some individuals in the population will die from competing causes of death, and are not potential victims of incremental radiation-induced cancer.

Each member of the hypothetical cohort is assumed to be exposed to a specified activity of a given radionuclide. In this analysis each member of the cohort annually inhales or ingests 1 pCi of the radionuclide, or is exposed to a constant external concentration of 1 pCi/cm^3 in air or 1 pCi/cm^2 on ground surfaces. Since the models used in RADRISK are linear, these results may be scaled to evaluate other exposure conditions. The cohort consists of an initial population of 100,000 persons, all of whom are simultaneously liveborn. In the scenario employed here, the radiation exposure is assumed to begin at birth and continue throughout the entire lifetime of each individual.

No member of the cohort lives more than 110 years. The span from 0 to 110 years is divided into nine age intervals, and dose rates to specified organs at the midpoints of the age intervals are used as estimates of the annual dose during the age interval. For a given organ, the incremental probability of death due to radiation-induced cancer is estimated for each year using radiation risk factors and the calculated doses during that year and relevant preceding years. The incremental probabilities of death are used in conjunction with the actuarial life tables to estimate the incremental number of radiation induced deaths each year.

The estimation of the number of premature deaths proceeds in the following manner. At the beginning of each year, m, there is a probability P^N of dying during that year from nonradiological causes, as calculated from the life table data, and an estimated incremental probability P^R of dying during that year due to radiation-induced cancer of the given organ. In general, for the m-th year, the calculations are:

$M(m)$ = total number of deaths in cohort during year m,

$= [P^N(m) + P^R(m)] \times N(m)$

$Q(m)$ = incremental number of deaths during year m due to radiation-induced cancer of a given organ,

$= P^R(m) \times N(m)$

$N(m+1)$ = number of survivors at the beginning of year m + 1

$= N(m) - M(m)$

P^R is assumed to be small relative to P^N, an assumption which is reasonable only for low-level exposures, such as those considered here (Bu81). The total number of incremental deaths for the cohort is then obtained by summing Q(m) over all organs for 110 years.

In addition to providing an estimate of the incremental number of deaths, the life table methodology can be used to estimate the total number of years of life lost to those dying of radiation-induced cancer, the average number of years of life lost per incremental mortality, and the decrease in the population's life expectancy. The total number of years of life lost to those dying of radiation-induced cancer is computed as the difference between the total number of years of life lived by the cohort assuming no incremental radiation risk, and the total number of years of life lived by the same cohort assuming the incremental risk from radiation. The decrease in the population's life expectancy can be calculated as the total years of life lost divided by the original cohort size (N(1)=100,000).

Either absolute or relative risk factors can be used. Absolute risk factors, given in terms of deaths per unit dose, are based on the assumption that there is some absolute number of deaths in a population exposed at a given age per unit of dose. Relative risk factors, the percentage increase in the ambient cancer death rate per unit dose, are based on the assumption that the annual rate of radiation-induced excess cancer deaths, due to a specific type of cancer, is proportional to the ambient rate of occurrence of fatal cancers of that type. Either the absolute or the relative risk factor is assumed to apply uniformly during a plateau period, beginning at the end of the latent period.

The estimates of incremental deaths in the cohort from chronic exposure are identically those which are obtained if a corresponding stationary population (i.e., a population in which equal numbers of persons are born and die in each year) is subjected to an acute radiation dose of the same magnitude. Since the total person-years lived by the cohort in this study is approximately 7.07 million, the estimates of incremental mortality in the cohort from chronic irradiation also apply to a one-year dose of the same magnitude to a population of this size, age distribution, and age-specific mortality rates. More precise life table estimates for a specific population can be obtained by altering the structure of the cohort to reflect the age distribution of a particular population at risk.

A.6 Risk Analysis Methodology

Risk estimates in current use at EPA are based on the 1980 report (BEIR-3) of the National Academy of Sciences Advisory Committee on the Biological Effects of Ionizing Radiation (NAS80). The form of these risk estimates is, to some extent, dictated by practical considerations, e.g., a desire to limit the number of cases which must be processed for each environmental analysis and a need to conform to limitations of the computer codes in use. For example, rather than analyze male and female populations separately, the risk estimates have been merged for use with the general population; rather than perform both an absolute and a relative risk calculation, average values have been used.

The derivation of the risk estimates from the BEIR-3 report is presented in Chapter 6. A brief outline of the general procedure is summarized below. Tables referenced from Chapter V of NAS80 are designated by a V prefix.

(1) The total number of premature cancer fatalities from lifetime exposure to 1 rad per year of low-LET radiation is constrained to be equal to the arithmetic average (280 per million person-rad) of the absolute and relative risk values (158 and 403 per million person-rad) given in Table V-25 of the BEIR-3 report for the L-L and L-L models for leukemia and solid cancers, respectively (NAS80).

(2) For cancers other than leukemia and bone cancer, the age and sex-specific incidence estimates given in Table V-14 were multiplied by the mortality/incidence ratios of Table V-15 and processed through the life table code at constant, lifetime dose rates of 1 rad per year. The resulting deaths are averaged, using the male/female birth ratio, and proportioned for deaths due to cancer in a specific organ as described in Chapter 6. These proportional risks are then used to allocate the organ risks among the 235.5 deaths per million person-rad remaining after the 44.5 leukemia and bone cancer fatalities (Table V-17) are subtracted from the arithmetic average of 280 given in Table V-25.

(3) The RADRISK code calculates dose rates for high- and low-LET radiations independently. A quality factor of 20 has been applied to all alpha doses to obtain the organ dose equivalent rates in rem per year (ICRP77). For high-LET radiation risk estimates, the risk from alpha particles is considered to be eight times that for low-LET radiation to the same tissue except for bone cancer, for which the risk coefficient is twenty times the low-LET value. Additional discussion was included in Chapter 6.

A typical environmental analysis requires that a large number of radionuclides and multiple exposure models be considered. The RADRISK code has been used to obtain estimates of cancer risk for unit intakes of approximately 200 radionuclides and unit external exposures by approximately 500 radionuclides. The calculated dose rates and mortality coefficients described in the preceding sections are processed through the life table subroutine of the RADRISK code to obtain lifetime risk estimates. At the low levels of contamination normally encountered in the environment, the life table population is not appreciably perturbed by the excess radiation deaths calculated and, since both the dose and risk models are linear, the unit exposure results may be scaled to reflect excess cancers due to the radionuclide concentrations predicted in the analysis of a specific source.

As noted in the discussion of the life table analysis, risk estimates for chronic irradiation of the cohort may also be applied to a stationary population having the same age-specific mortality rates as the 1970 U.S.

population. This is, since the stationary population is formed by superposition of all age groups in the cohort, each age group corresponds to a segment of the stationary population with the total population equal to the sum of all the age groups. Therefore, the number of excess fatal cancers calculated for lifetime exposure of the cohort at a constant dose rate would be numerically equal to that calculated for the stationary population exposed to an annual dose of the same magnitude. Thus, the risk estimates may be reported as a lifetime risk (the cohort interpretation) or as the risk ensuing from an annual exposure to the stationary population. This equivalence is particularly useful in analyzing acute population exposures. For example, estimates for a stationary population exposed to annual doses which vary from year to year may be obtained by summing the results of a series of cohort calculations at various annual dose rates.

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APPENDIX B

GLOSSARY AND ACRONYMS

GLOSSARY

actinide:	The series of elements beginning with actinium, Element No. 89, and continuing through lawrencium, Element No. 103.
alpha particle:	Positively charged particle emitted by certain radioactive materials. It is made up of two neutrons and two protons, identical to the nucleus of a helium atom. It is the least penetrating type of radiation.
beta particle:	An elementary particle emitted from a nucleus during radioactive decay, with a single electrical charge and a mass equal to 1/1837 that of a proton. A negatively-charged beta particle is identical to an electron. A positively-charged beta particle is called a positron.
contact-handled TRU wastes:	TRU wastes that can be handled with just the shielding that is provided by the waste package itself.
critical organ:	Specific organ being most susceptible to the effects of a specific type of radiation.
Ci:	Curie - the unit rate of radioactive decay; the quantity of any radionuclide which undergoes 3.7×10^{10} disintegrations/second. Several fractions of the curie are in common usage: Nanocurie (nCi) - one-billionth of a curie; 3.7×10^{1} disintegrations/second Microcurie (μCi) - one-millionth of a curie; 3.7×10^{4} disintegrations/second Millicurie (mCi) - one-thousandth of a curie; 3.7×10^{7} disintegrations/second Picocurie (pCi) - one-millionth of a microcurie; 3.7×10^{-2} disintegrations/second or 2.22 disintegrations/minute
daughter:	Synonym for decay product.

decay product:	A nuclide resulting from the radioactive disintegration of a radionuclide, being formed either directly or as the result of successive transformations in a radioactive series. Also called a daughter. Decay products may be stable or radioactive.
dose:	The amount of energy absorbed per gram of absorbing tissue as a result of the exposure.
dose equivalent:	A term used to express the amount of effective radiation when modifying factors have been considered; the product of absorbed dose multiplied by a quality factor multiplied by a distribution factor. It is expressed numerically in rems.
dosimetry:	Quantification of energy absorbed by the population from decaying radionuclides.
effective half-life ($t_{\frac{1}{2}}$):	The time required for one-half of a radioactive material originally present in the body to be removed by biological clearance or radioactive decay.
fissile:	Any material fissionable by neutrons of all energies, including thermal (slow) neutrons as well as fast neutrons.
fission:	The splitting of a heavy nucleus into approximately equal parts (which are nuclei of lighter elements), accompanied by the release of a relatively large amount of energy. Fission can occur spontaneously, but usually is caused by nuclear absorption of gamma rays, neutrons, or other particles.
fission products:	The nuclei formed by the fission of heavy elements, plus the nuclides formed by the fission fragments' radioactive decay.
fuel cycle:	The series of steps involved in supplying fuel for nuclear power reactors. It includes mining, refining, the original fabrication of fuel elements, their use in a reactor, chemical processing to recover the fissionable material remaining in the spent fuel, re-enrichment of the fuel material, and refabrication into new fuel elements.
gamma ray:	High-energy, short-wavelength electromagnetic radiation. Gamma radiation frequently accompanies alpha and beta emissions and always accompanies fission. Gamma rays are very penetrating, and are best stopped by dense materials.

general environment:	The total terrestrial, atmospheric, and aquatic environments outside sites within which any activity, operation, or process associated with the management and storage of spent nuclear fuel, high-level, or transuranic radioactive wastes is conducted.
geometric mean:	The mean of a set of numbers, calculated by taking the Nth root of the product of N numbers or by finding the arithmetic mean of the logarithms of the individual numbers.
geometric standard deviation:	The standard deviation of a set of numbers obtained when calculating the arithmetic mean of the logarithm of the individual numbers. Also see geometric mean.
geosphere:	The solid portion of the earth, synonomous with the lithosphere.
GW:	Gigawatts = one billion (10^9) watts.
heavy metal:	All uranium, plutonium, or thorium placed into a nuclear reactor.
high-level radioactive waste:	Waste whose radioactivity is predominantly characterized by high-energy radiation; consists of the byproducts of nuclear reactors and wastes generated by spent fuel processing operations of the nuclear fuel cycle. These are highly radioactive materials resulting from the reprocessing of spent nuclear fuel, including liquid waste produced directly in reprocessing and any solid material derived from such liquid waste.
high-temperature gas-cooled reactor:	Nuclear reactor using uranium and thorium as a fuel whose core is designed for high fuel utilization efficiency. The heat removal system is based upon helium as a coolant.
ionizing radiation:	Any electromagnetic or particulate radiation capable of producing ions, directly or indirectly, in its passage through matter.
irregularly inherited disorders:	Genetic conditions with complex causes, constitutional and degenerative diseases, etc.

isotope:	One of two or more atoms with the same atomic number (the same chemical element) but with different atomic weights. Isotopes usually have very nearly the same chemical properties, but some have somewhat different physical properties.
kg:	Kilogram - the SI unit of mass, approximately equal to 2.2 pounds.
light-water reactor (LWR):	A nuclear reactor whose heat removal system is based on the use of ordinary water as the moderator and reactor coolant.
linear energy transfer (LET):	The rate at which charged particles transfer their energy to the atoms in a medium; expressed as energy lost per distance traveled in the medium.
lognormal distribution:	A distribution of the frequency of a value plotted on a linear scale versus the value plotted on a logarithmic scale, which results in a bell-shaped curve.
m^3:	Cubic meter - the SI unit of volume, approximately equal to 35.3 cubic feet.
management and storage:	Any activity, operation, or process, except for transportation, conducted to prepare spent nuclear fuel, high-level or transuranic radioactive wastes for storage or disposal, the storage of any of these materials, or activities associated with disposal of these wastes.
member of the public:	Any individual who is not engaged in operations involving the management, storage, and disposal of materials covered by these standards. A worker so engaged is a member of the public except when on duty at a site.
metric ton (t):	The SI unit of weight equal to 1000 kilograms or 2205 pounds.
mR/h:	See Roentgen.
nanocurie:	See curie.
neutron activation:	The process of making a material radioactive by bombardment with neutrons.

neutron capture:	The process in which an atomic nucleus absorbs or captures a neutron. The probability that a given material will capture neutrons is dependent on the energy of the neutrons and on the nature of the material.
neutron:	An uncharged elementary particle with a mass slightly greater than that of a proton, and found in the nucleus of every atom heavier than hydrogen. Neutrons sustain the fission chain reaction in a nuclear reactor.
noble gas:	Any of a group of rare gases that include helium, neon, argon, krypton, xenon, and sometimes radon and exhibit great stability and extremely low reaction rates.
nonstochastic effect:	Those health effects that increase in severity with increasing dose and usually have a threshold.
rad (radiation adsorbed dose):	A measure of the energy imparted to matter by radiation; defined as 100 ergs per gram.
radioactive decay:	A process whereby the nucleus of an atom emits excess energy. The emission of this energy is referred to as radioactivity.
radioactivity:	The property of certain nuclides of spontaneously emitting particles or gamma radiation or of emitting X-radiation following orbital electron capture or of undergoing spontaneous fission.
radionuclide:	A radioactive nuclide.
RBE:	The ratio of the dose (rad) of low-LET radiation to the does of high-LET radiation producing the same endpoint. It is a measure of the effectiveness of high-LET compared to low-LET radiation in causing the same specific endpoint.
	Millirad (mrad) - one thousandth of a rad.
rem (roentgen equivalent man):	A measure of equivalence for the relative biological effect of radiations of different types and energies on man.
remotely-handled TRU waste:	Those types of TRU wastes that must be handled by robotics.

risk projection:	Absolute - risk projection based on the assumption that there is some absolute number of deaths in a population exposed at a given age per unit of dose. Relative - risk projection based on the assumption that the annual rate of radiation-induced excess cancer deaths is proportional to the ambient rate of occurrence of fatal cancer.
roentgen:	R is the symbol for roentgen, a unit of measurement of X-radiation, equivalent to an absorbed dose in tissue of approximately 0.9 rad. Milliroentgen (mR/h) - one-thousandth of a roentgen.
spent nuclear fuel:	Any nuclear fuel removed from a nuclear reactor after it has been irradiated and whose constituent elements have not been separated by reprocessing.
standards:	The "limits" on radiation exposures or levels, or concentrations or quantities of radioactive material, in the general environment outside the boundaries of locations under the control of persons possessing or using radioactive material.
stochastic effect:	Those health effects for which the probability of occurrence is a function of the dose received.
storage:	Placement of radioactive wastes with planned capability to readily retrieve such materials.
target:	Material subjected to particle bombardment or irradiation in order to induce a nuclear reaction.
target theory (Hit theory):	A theory explaining some biological effects of radiation on the basis that ionization occurring in a discreet volume (the target) within the cell, directly causes a lesion which subsequently results in a physiological response to the damage at that location. One, two, or more "hits" (ionizing events within the target) may be necessary to elicit the response.
teratogenesis:	Congenital abnormalities or defects.
transuranic waste:	Waste containing more than 100 nanocuries of alpha-emitting transuranic isotopes, with half-lives greater than 20 years, per gram of waste.

X-ray:	Penetrating electromagnetic radiation whose wave lengths are shorter than those of visible light. They are usually produced by bombarding a metallic target with fast electrons in a high vacuum. In nuclear reactions, it is customary to refer to photons originating in the nucleus as gamma rays, and those originating in the extranuclear part of the atom as X-rays. These rays are sometimes called roentgen rays.
Zircaloy:	A zirconium alloy used as fuel cladding in some types of nuclear reactors.

ACRONYMS

AEC	U.S. Atomic Energy Commission
ALAP	As low as practicable
ALARA	As low as reasonably achievable
AMAD	Activity median aerodynamic diameter
ANL	Argonne National laboratory
BEAR	Biological Effects of Atomic Radiation
BEIR	Biological Effects of Ionizing Radiation
BID	Background Information Document
CFR	Code of Federal Regulations
CH	Contact-handled
CRWM	Civilian Radioactive Waste Management
DEIS	Draft Environmental Impact Statement
DOD	U.S. Department of Defense
DOE	U.S. Department of Energy
DOT	U.S. Department of Transportation
DREF	Dose rate effectiveness factor
DWPF	Defense Waste Processing Facility
ERC	President's Federal Energy Resources Council
ERDA	Energy Research and Development Administration
EPA	U.S. Environmental Protection Agency
FFTF	Fast Flux Test Facility
FRC	Federal Radiation Council

GI	Gastrointestinal
GW(e)	Gigawatts of electric power
HANF	Hanford, Washington
HEW	U.S. Department of Health, Education, and Welfare
HLW	High-level radioactive waste
HTGR	High-temperature gas-cooled reactor
ICPP	Idaho Chemical Processing Plant
ICRP	International Commission on Radiological Protection
ICRPTG	International Commission on Radiological Protection Task Group
INEL	Idaho National Engineering Laboratory
IRG	Interagency Review Group
LANL	Los Alamos National Laboratory
LET	Linear energy transfer
LLI	Lower large intestine
LMFBR	Liquid metal fast breeder reactor
LQ	Linear quadratic
LWR	Light-water reactor
MFRP	Midwest Fuel Recovery Plant
MIRD	Medical internal radiation dose
MRS	Monitored retrievable storage
MTHM	Metric tons of heavy metal
NCHS	National Center for Health Statistics
NCRP	National Council on Radiation Protection and Measurements
NFS	Nuclear Fuel Services
N-P	Nasophoryngial
NRC	U.S. Nuclear Regulatory Commission

NRPB	National Radiological Protection Board
NWPA	Nuclear Waste Policy Act of 1982
NWTS	National Waste Terminal Storage
OMB	Office of Management and Budget
ORNL	Oak Ridge National Laboratory
P	Pulmonary
R	Roentgen
RBE	Relative biological effectiveness
RFP	Rocky Flats Plant
RH	Remote-handled
RIA	Regulatory Impact Analysis
S	Stomach
SAB	Science Advisory Board
SI	Small intestine
SRP	Savannah River Plant
T-B	Tracheobronchial
TRU	Transuranic
ULI	Upper large intestine
UNSCEAR	United Nations Scientific Committee on the Effects of Atomic Radiation
WIPP	Waste Isolation Pilot Plant

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